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VOLUME II

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CHAPTER XII

THE MORTALITIES OF INFANCY

By Richard Arthur Bolt, M.D., Gr.P.H.,
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Infant Mortality. A Symptom-complex.—Infant mortality is a symptom-complex whose diverse factors—hereditary, congenital and environmental—when judiciously balanced and evaluated present a composite picture of community life of absorbing interest and practical socio-medical value. The mortalities of infancy traced to their finer ramifications reveal not only the sanitary status of a community but its social, economic and moral aspects as well. By a respectable, and ever enlarging, group of public health workers interested in child hygiene the infant mortality rate has come to be regarded as the most sensitive index we have of social and sanitary progress. Thus, Sir Arthur Newsholme speaking out of his wealth of experience states that "infant mortality is the most sensitive index we possess of social welfare and of sanitary administration, especially under urban conditions."*

The study and prevention of infant mortality has commanded the attention of philanthropic individuals and health authorities for some time, but its greatest popular impetus has come since the opening of the twentieth century. The social significance of infant mortality was early recognized. In his preface to "Infant Mortality—A Social Problem," Sir George Newman says "it cannot be too distinctly recognized, as Sir John Simon pointed out, that a high infant mortality rate almost necessarily denotes a prevalence of those causes and conditions which in the long run determine a degeneration of race; and further, that a high death-rate of infants is an indication of the existence of evil conditions in the homes of the people—which are, after all, the vitals of the nation."† Growing out of careful and extensive studies, fortified by the accumulated experience of many communities where intensive child hygiene work has been carried on, there has come the conviction that infant mortality can, and should be, largely prevented. No longer can "man's inhumanity to man," as exemplified too often by his neglect and cruelty to children, be condoned by the antiquated notion that "God sets the infant mortality rate." The causes of infant morbidity and mortality are largely preventable and man himself can do much if he will to mitigate or prevent them.

---

* Local Government Board of England and Wales, Thirty-ninth Annual Report, 1909–1910, Supplement to the Report of the Board's Medical Officer, Report on Infant and Child Mortality (Cd. 5263), p. 74.

† Newman, Sir George, Infant Mortality—A Social Problem, Preface, p. v., E. P. Dutton and Co., New York City (1907).

The Eugenic Argument.—There are on the other hand a skeptical group, largely followers of the Karl Pearson School of Biometrics, who join hands with certain of the Eugenists in urging that "Nature, not nurture" is the determining factor in infant mortality. They confidently state that on the whole a reduction of infant mortality is not an unmixed benefit to society, but tends to obstruct Nature in her struggle for existence by perpetuating the "weaklings" and "unfit." Popenoe and Johnson put it coldly when they say that "part of the children born in any district in a given year are doomed by heredity to an early death; that if they die in one year they will not be alive to die in the succeeding year and *vice versa*."* To this school of thought it is evident that further reduction of infant mortality is problematic and questionable as to eugenic results.†

The argument for this group of workers has been summed up by Popenoe and Johnson as follows: "We admit that it is possible to keep a lot of children alive who would otherwise have died in the first few months of life. It is being done, as the New York figures, and pages of others that could be cited, prove. The ultimate result is twofold: some of those who are doomed by heredity to a selective death, but are kept alive through the first year, die in the second or third or fourth year. They must die sooner or later; they have not inherited sufficient resistance to survive more than a limited time. If they are by great effort carried through the first year, it is only to die in the next. This is a statement which we have nowhere observed in the propaganda of the infant mortality movement; and it is perhaps a disconcerting one. It can only be proved by refined statistical methods, but several independent determinations by the English biometricians leave no doubt as to the fact."‡ The work of Karl Pearson, E. C. Snow and Ethel M. Elderton is cited to substantiate this statement. "There is still another consequence," they continue. "If the gain is by great exertions made more than temporary; if the baby who would otherwise have died in the first months is brought to adult life and reproduction, it means in many cases the dissemination of another strain of weak heredity, which natural selection would have cut off ruthlessly in the interests of race betterment. In so far, then, as the infant mortality movement is not futile it is, from a strict biological viewpoint, often detrimental to the future of the race."§

Influence of Infant Mortality on Mortality at Higher Ages.—These and similar arguments have been challenged and, in the opinion of the writer, amply answered by those who have had not only the special statistical training but also actual practical administrative experience in the reduction of infant mortality. The eminent public health authority, Sir Arthur Newsholme, has shown in a series of classical reports to the Local Government Board of England that "a high infant death-rate in a given community implies in general a high death-rate in the next four years of life, while low death-rates at both age periods are similarly associated." In other words there is a "very high correlation between the amount of infant mortality and of mortality at ages one to five."*

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* Applied Eugenics by Popenoe, Paul and Johnson, Roswell Hill, p. 413. The Macmillan Company, New York (1918).

† *Ibid.*, pp. 414–417, also, Greenwood, M. and Brown, J. W., An Examination of Some Factors Influencing the Rate of Infant Mortality, Jour. Hyg., xii (1912), pp. 5–45, also, Pearson, Prof. Karl and Elderton, Miss Ethel M., The Relative Strength of Nurture and Nature, 2d Edit. London (1915).

‡ Applied Eugenics by Popenoe, Paul and Johnson, Roswell Hill, p. 413.

§ *Ibid.*, p. 414.

Sir Arthur is very firm in his conviction that "there need therefore be no hesitation in making every practicable effort to reduce infant mortality. It is not only in accord with the highest feelings of humanity, but action which secures reduction of infant mortality also secures reduction of mortality at higher ages."† Holt, who has been intimately associated with the child hygiene movement in the United States since its beginning, is equally convinced that "a high infant mortality is in no sense a protection to our body politic. We must eliminate the unfit by *birth*, not by death. The race is to be most effectively improved by preventing marriage and reproduction by the unfit, among whom we would class the diseased, the degenerate, the defective and the criminal. In working for the survival of the feeble and the unfortunate we are not contravening nature's law and striving to save the unfit. A high infant mortality results in a sacrifice of the *unfortunate*, not the *unfit*."‡

No large community in the United States has shown more clearly what could be accomplished by intensive and well directed child hygiene work in reducing infant mortality than has New York City. The average infant mortality rate during the years 1911–1915 was 102. In 1917 the rate had dropped to 88.8, in 1919 it was 81.6 and in 1921 71.0. In commenting upon this before the Senate Committee considering the "Sheppard-Towner Bill for the Public Protection of Maternity and Infancy," Dr. S. Josephine Baker, Chief of the Bureau of Child Hygiene of New York City, said, "One of the great objections that has been raised against any intensive child welfare work has been that it perpetuates the unfit. In order to meet this objection we have kept rather careful statistics in New York City with regard to the child after it has passed the age of one year. Five years after we began our work with babies we began to watch the effect upon the mortality of children under five years of age. We have found since that time that the mortality in this age group—under five years—has shown a decline that has been much greater than that in the group under one year of age. In other words, the children are not only alive and well at the end of their first year, but they continue in good health throughout child life."*

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* Local Government Board of England and Wales, 39th Annual Report, 1909–10 (Cd. 5263), p. 13.

† *Ibid.*, p. 17.

‡ Holt, L. Emmett, Infant Mortality, Ancient and Modern; An Historical Sketch. Presidential Address at the Fourth Annual Meeting of the American Association for the Study and Prevention of Infant Mortality. Transactions of the A. A. S. P. I. M. (1913), p. 25.

TABLE 1.—SHOWING THAT THE EFFECT OF IMPROVED CARE DURING INFANCY IS NOTICEABLE ALSO IN AN INCREASED RESISTANCE TO DISEASE DURING CHILDHOOD

Based on figures from the New York City Bureau of Child Hygiene, 1904–1919. Baker, S. Josephine, The Bureau of Child Hygiene, New York City Department of Health, 3d ed., N. Y., 1915, Monograph series No. 4 and information from the Bureau of Child Hygiene in 1920 compiled by Ernst Christopher Meyer, in Infant Mortality in New York City, Publication No. 10 of the Rockefeller Foundation, International Health Board, New York City (1921), p. 128, Table III.

(*Death-rates per* 1000 *Living Children in New York City*)

Year	Under one year	Between		Under five years
		One and two years	Two and five years	
1904	164.0	56.9	17.9	58.5
1905	163.0	47.2	14.7	54.9
1906	164.0	50.0	14.8	56.2
1907	160.0	46.1	14.2	54.9
1908	144.0	41.9	13.1	50.0
1909	137.0	45.3	13.1	49.5
1910	134.0	40.1	12.2	47.7
1911	120.0	34.4	10.5	42.1
1912	110.0	31.1	9.4	38.3
1913	102.0	30.5	9.5	36.4
1914	94.6	29.8	9.2	32.9
1915	98.2	29.1	8.9	
1916	93.1	28.3	10.9	
1917	88.8	23.9	7.8	
1918	91.7	32.8	11.8	
1919	81.6	....	6.9	
Average Rate for Five-year Periods				
1904–08 incl.	159.0	48.4	15.0	54.9
1909–13 incl.	120.0	36.3	10.9	42.8
1914–18 incl.	93.2	28.4	9.7	
Decrease per cent. 1904–1913	24.5	25.0	27.0	22.0
Decrease per cent. 1914–1918	22.3	21.7	11.0	

NOTE.—1914–1919 rates under one year of age based on reported births; all other rates based on estimated population.

In our enthusiasm concerning the reduction of infant mortality during recent years, we should not overlook the important fact that many of the untoward conditions which determine a high infant mortality are also prejudicial to life at all ages, although acting with greater force in the earlier years. The considerable reduction in infant mortality has undoubtedly reflected improvements in general sanitary measures and better social and economic conditions, as well as a gradually increasing intelligence on the part of mothers regarding infant hygiene. Any specific measures for the reduction of infant mortality must always be balanced with all these other factors. Infant hygiene cannot be set upon a pedestal by itself but must be integrated with all other efforts for child welfare in the community.

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* Hearing before the Committee on Public Health and National Quarantine, United States Senate, Sixty-sixth Congress, Second Session, Senate Bill 3259 for the Public Protection of Maternity and Infancy, p. 49. Washington, D. C., 1920.

Infant Mortality and Damage Rates among Survivors.—There is another aspect of the problem which should be carefully considered. By throwing all possible safeguards about the pregnant mother and by applying well tested methods of prenatal care and infant hygiene we not merely save the life of the child throughout the first year, but may prevent the development of many conditions which leave more or less serious defects. There is no question that a high infant mortality goes apace with a high damage rate among those who survive. While the mortality rate in the years succeeding infancy is comparatively low, the damage rate is very high. We are just beginning to appreciate the supreme importance of the pre-school age in its bearing upon the future health of the child. In this period arise many of the defects from which the child later suffers. Infant mortality, therefore, is not only an index of existing conditions; it is at once a reflection upon the past and a prophecy of the childhood of tomorrow. The initial casualites of war are long forgotten, or have become enshrined memories, while we are still caring for our *damaged* soldiers.

Influence of Infant Mortality on General Death-rate.—A careful analysis of the vital statistics of those countries where complete and reliable returns have been compiled reveals that the reduction of infant mortality has been one of the prime factors in lessening the general death-rate. At the present time a substantial reduction in the general death-rate rests largely in still further cutting down infant mortality, especially in the early weeks of life. In England and Wales from 1841 to 1870 inclusive the annual standardized death-rates at all ages ranged from 20.6 to 22.4 per 1000 living, and the infant mortality rates from 148 to 160 deaths under one year per 1000 live births. Very little change in a downward trend is noted either in the general mortality or in the infant mortality rates. About 1870 a distinct reduction in the general death-rate began which, with yearly fluctuations, has kept declining ever since, reaching the remarkably low rate of 12.4 in 1920. Infant mortality did not begin to show a marked reduction until some years later and not until after 1910 did the precipitate drop occur which reached 89 deaths under one year of age per 1,000 live births in 1919* and the surprising figure of 80 in 1920.

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* Eighty-second Annual Report of the Registrar General of Births, Deaths and Marriages in England and Wales (1919), Table 8, p. 9 (cmd. 1017), London (1920).

It is reasonable to infer that general sanitary improvements in England after 1870 first began to be reflected in the general mortality rates, but had very little if any influence upon the infant mortality rates until intensive efforts for child hygiene had been put forth. When community organization and direct measures for the prevention of infant mortality, largely initiated by voluntary societies, came under government sanction and support, considerable impetus was given to child hygiene work. This has had a decided cumulative effect in still further reducing the general mortality.

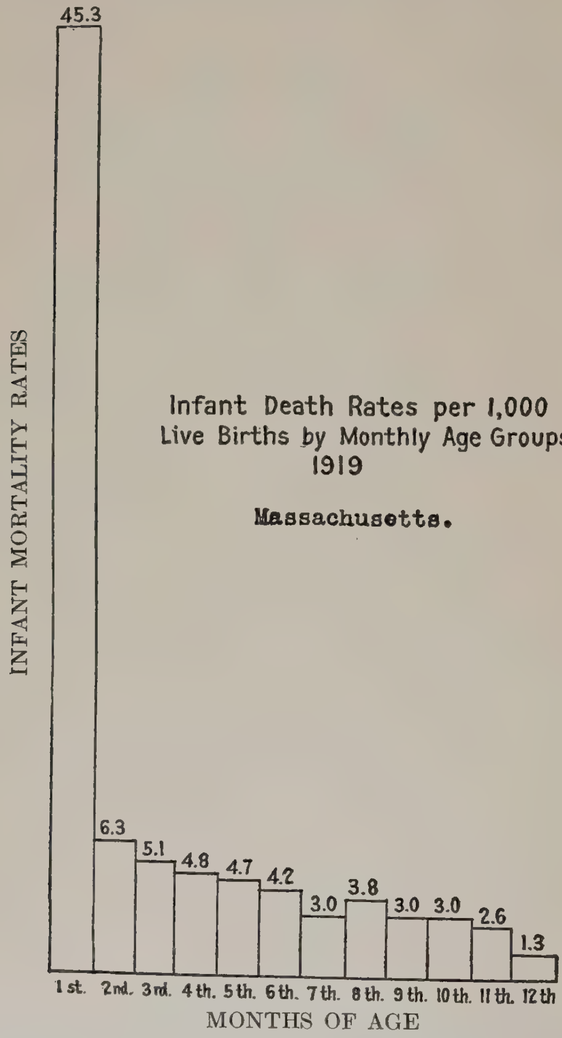

Fig. 1.—From the report of a special commission to investigate maternity benefits. Boston, Massachusetts, December, 1920. House Report No. 1835, p. 55.

The same tendency has been noted in the United States and other countries where intensive campaigns for the reduction of infant mortality have stimulated local authorities to study their problems and devise suitable means of solving them.

Gain and Loss in Infant Lives.—The reduction in the mortalities of infancy thus far effected has taken place largely in the latter half of the first year of life. Very little, if any, progress has been made in limiting deaths in the neonatal period. Upward of 40 per cent. of the deaths during the first year of life occur in the first month. In Massachusetts during 1919 the infant mortality rate was 88.2. Over 50 per cent. of these deaths (or 45.3 per 1000 live births) occurred before the babies had reached one month of age. On a conservative basis 40 per cent. of the neonatal deaths could have been prevented by proper prenatal and obstetrical care. Figures 1 and 2 show graphically the age distribution of deaths under one year in Massachusetts during 1919. Note particularly the high mortality in the first month and in the first few days of life. These charts are typical of what might be shown for other localities throughout the United States.

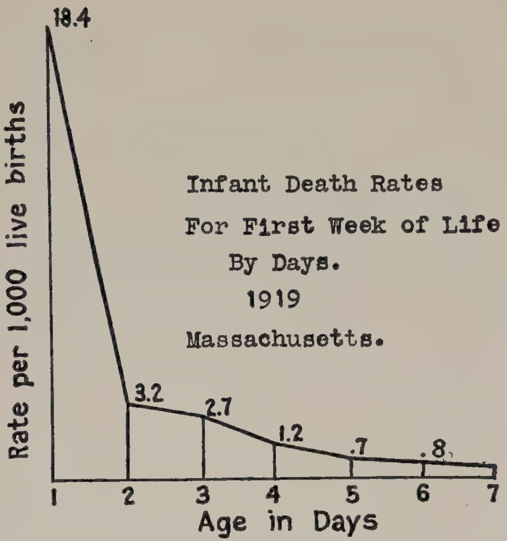

FIG. 2.—From the report of a special commission to investigate maternity benefits. Boston, Massachusetts, 1920. House Report No. 1835, p. 54.

Maternal Mortality.—A shockingly large number of mothers are still sacrificed each year. The rates in the United States are uniformly higher than in a number of foreign countries. The maternal mortality rate has been gradually advancing in recent years. It is estimated that an increase of 15 per cent. took place in the first six months of

1920 as compared with 1919.* Hand in hand with this, the number of abortions and still births remains abnormally high.

TABLE 2.—MATERNAL MORTALITY RATES IN CERTAIN FOREIGN COUNTRIES COMPARED WITH THE UNITED STATES BIRTH REGISTRATION AREA PER 1000 BIRTHS

Rates from the latest available official reports up to 1919

Compiled by Robert M. Woodbury, Statistician, U. S. Children's Bureau, Washington, D. C.

Australia (1918)	4.7
England and Wales (1919)	4.4
Finland (1916)	3.6
France (1913)	4.6
Hungary (1915)	4.0
Ireland (1918)	4.8
Italy (1915)	2.2
Japan (1916)	3.5
New Zealand (1919)	5.1
Norway (1915)	2.7
Prussia (1914)	3.5
Scotland (1919)	6.2
Spain (1915)	5.2
Sweden (1915)	2.9
Switzerland (1915)	5.5
The Netherlands (1919)	3.4
United States (Birth Registration Area, 1919)	7.4

MATERNAL MORTALITY RATES, 1915–1920. U. S. BIRTH REGISTRATION AREA

Year	Deaths from puerperal causes per 1000 live births		
	All puerperal causes	Puerperal septicemia	All other puerperal causes
1915	6.1	2.4	3.7
1916	6.2	2.5	3.7
1917	6.6	2.7	3.9
1918	9.2	2.6	6.6
1919	7.4	2.5	4.9
1920	8.0	2.7	5.3

While it is true that sanitation and infant hygiene have had a marked effect in reducing infant mortality in the sixth, seventh and eighth months of life they have not made a dent upon the birth mortalities, nor influenced perceptibly the miscarriages and still births. It is clear that prenatal and neonatal causes of death may continue to act throughout the first year of life and, although they rapidly decrease in intensity during the following four years, their influence may still be noted at later periods. *Apropos* of this, Sir Arthur

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* Statistical Bulletin, Metropolitan Life Insurance Co., Louis I. Dublin, Statistician, vol. i, No. 2 (Nov., 1920), p. 8.

Newsholme has pointed out the "immense national importance of reduction of infant mortality," even in the earliest months, "for an excessive infant mortality is nearly always associated with an excessive death-rate during the next four years of life, and communities showing excessive death-rates in the first five years of life suffer to a greater or less extent from excessive death-rates right up to adult life. Conversely, low infant mortalities are associated in the same communities with low death-rates at subsequent ages up to adult life; and there can be little doubt that if the influence of migration of population could be eliminated, the effect of conditions influencing health during the early years of life would be found to persist throughout adult life. Nor are these effects restricted to deaths. All evidence points to the conclusion that the general standard of health is higher in districts having a low mortality in early life."* In order to show the interrelation of some of these factors a table is inserted giving the English experience over a series of years.

ENGLAND AND WALES

TABLE 3.—Compiled by Dr. Janet M. Campbell, Senior Medical Officer for Maternity and Child Welfare, Ministry of Health, and presented in an address on "Maternity Homes" before the Second English Speaking Conference on Infant Welfare, London, July, 1921.

Year	Birth-rate	Death-rate	Infant mortality rate	Maternal mortality (Classification as in use before 1911)			
				No. of deaths	Deaths per 1000 births		
					Sepsis	Other causes	Total childbirth
1905	27.3	15.3	128	3905	1.87	2.33	4.20
1906	27.2	15.5	132	3757	1.75	2.27	4.02
1907	26.5	15.1	118	3520	1.60	2.23	3.83
1908	26.7	14.8	120	3361	1.48	2.09	3.57
1909	25.8	14.6	109	3379	1.56	2.14	3.70
1910	25.1	13.5	105	3191	1.42	2.14	3.56
1911	24.3	14.6	130	3236	1.52	2.15	3.67
1912	23.9	13.3	95	3299	1.47	2.31	3.78
1913	24.1	13.8	108	3271	1.34	2.37	3.71
1914	23.8	14.0	105	3469	1.63	2.32	3.95
1915	21.9	15.7	110	3210	1.56	2.38	3.94
1916	20.9	14.4	91	3038	1.47	2.40	3.87
1917	17.8	14.4	96	2446	1.39	2.27	3.66
1918	17.7	17.6	97	2353	1.35	2.20	3.55
1919	18.5	13.8	89	2852	1.76	2.36	4.12
1920	25.4	12.4	80	3942	1.87	2.25	4.12

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* Local Government Board of England and Wales, 39th Annual Report, 1909–10 (Cd. 5263), p. 19.

The Appeal to the Medical Man.—To the medical man the problems of infant mortality make more than a professional appeal. In their study he is inevitably brought face to face with socio-economic forces which not only influence the life of his own community, but also affect the future of medicine itself. His social conscience is quickened and his humanitarian interests activated. The physician, whether a pediatrist or not, cannot escape the responsibility which faces him in a community where infant mortality remains unduly high. He above all others should be in a position to point out the various factors entering into the infant mortality problem and take definite steps to assist his community in gearing up their resources to bring about a substantial reduction.

The treatment and cure of the comparatively small number of ill babies which come under the surveillance of any one physician in his private practice year by year can influence but little the infant mortality rate. It is therefore unbecoming, as well as untenable, for any physician to take the position that organized child hygiene work in his community will seriously cut into his private practice. As a matter of fact, in those communities where infant hygiene work has been well conceived and carried out with the hearty coöperation of the medical profession it has proved satisfactory to physicians and mothers alike in making it possible to get the babies at a much earlier period so as to forestall their sickness, or institute treatment at a time when the best results can be obtained. The mothers themselves have soon learned the lesson to consult a physician early. It has also made it possible through trained public health nurses for the physician to secure assistance in carrying out the details of weighing and measuring the baby and of seeing that his directions are carefully followed in the home.

Variability of Infant Mortality.—The most outstanding feature of infant mortality is its variability. (See Table 4, showing infant mortality rates in various countries.) It exhibits marked geographical, racial, seasonal and social variations. The infant mortality rate varies markedly not only in different parts of the same country, but in neighboring cities and adjacent wards of the same city and in the same country in different years. The factors entering into it are so complex and interwoven that no one formula can be applied for its complete solution. Each factor must be weighed and studied in its proper relationship. Intensive methods of infant hygiene must be applied to reduce the mortality factor by factor until the lowest possible denominator, as it were, has been obtained.

General Classification of Causes of Infant Mortality.—

1. Congenital diseases. (Injuries at birth and diseases of early infancy are classed under a variety of indefinite terms, such as atrophy, marasmus, debility, etc.)
2. Gastro-intestinal diseases.
3. Respiratory diseases.
4. Acute infectious diseases of childhood.
5. Other causes.

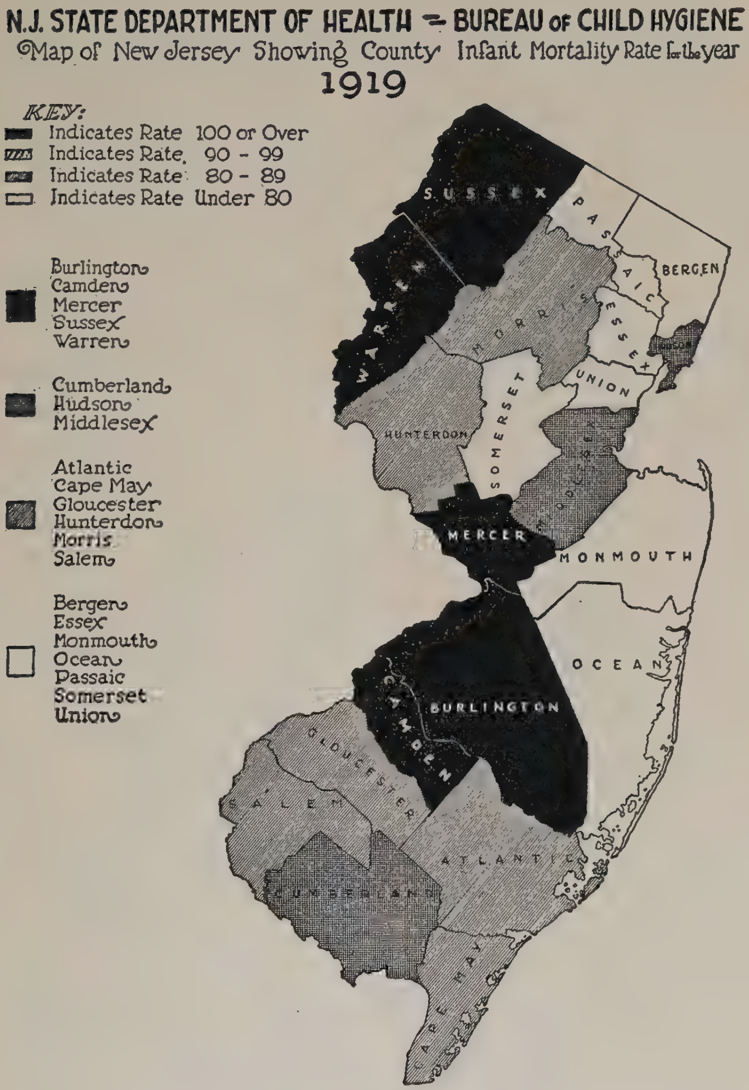


FIG. 3.—Local variability of infant mortality in New Jersey.

TABLE 4.—SHOWING INFANT MORTALITY RATES IN VARIOUS FOREIGN COUNTRIES IN COMPARISON WITH THE BIRTH REGISTRATION AREA OF U. S. A.

By sex

Excerpts from Birth Statistics for the Birth Registration Area of the United States for 1919, Bureau of the Census, page 31.

Country	Year	Infant mortality rates (Deaths under one year per 1000 births)		
		Male	Female	Total
United States Registration Area	1919	95.8	77.0	86.6
Australian Commonwealth	1919	76.3	61.7	69.0
Ceylon	1919	227.8	217.3	222.5
Chile	1918	260.9	248.2	254.5
Denmark	1918	81.8	65.6	73.7
England and Wales	1919	100.0	77.6	89.0
France	1919	.....	.....	119.0
Germany* (excluding Alsace-Lorraine)	1918	167.0	140.0	154.0
Ireland	1918	95.7	76.6	86.1
Italy	1916	174.5	157.7	166.1
Jamaica	1919	167.7	155.4	161.5
Japan	1916	178.9	161.2	170.0
The Netherlands	1917	63.6	49.9	56.7
New Zealand	1918	53.6	43.0	48.3
Spain	1915	165.7	145.3	155.5
Sweden	1915	83.8	67.4	75.6
Switzerland	1918	96.9	79.1	88.0

* Delbrück, Stat. Reichsamt, Berlin.

Data supplied by Department of Vital Statistics, League of Red Cross Societies, Geneva, Knud Stuman, Statistician (1921).

The death-rates per 1000 estimated midyear population under one year from the important causes which make up this broad classification for the years 1910–1918 in the Registration Area for Deaths in the United States are given in Table 5. It is instructive to compare this table with Table 6 showing the per cent. of deaths from certain important causes to total deaths under one year of age in the Registration Area as of 1910 in the United States from 1910 to 1919 inclusive. Figure 4 shows graphically the infant death rates for the principal causes in Massachusetts for the year 1919.

The percentage of deaths under one year of age to total deaths in the United States has gradually decreased from 19.4 in 1910 to 14.4 in 1919. A study of the classified subdivisions of the first year of life for the period 1910–1919 reveals that there has been practically no decrease under one month (Table 7). The greatest decrease has occurred in the periods three to five months and six to eight months. A glance at Table 5 will make it clear that there has been a con-

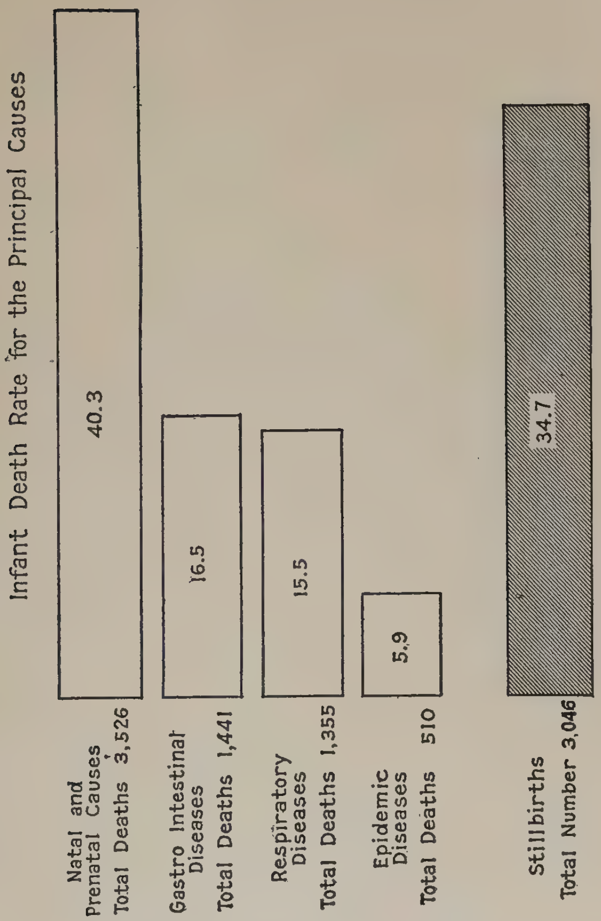


FIG. 4.—Massachusetts House Report 1920, No. 1835, p. 53.

siderable increase in deaths from "premature births," "malformations" and "injuries at birth" while the most marked *decrease* has occurred in "diarrhea and enteritis." The increase in "premature births" may be only apparent, however, on account of a transference in the cause of death.

Table 5.—Death-rate per 1000 Estimated Midyear Population Under One Year of Age from Certain Important Causes in the Registration States as of 1910: (Exclusive of North Carolina) 1910 to 1918

Abridged int. List No.	Cause of death	1918	1917	1916	1915	1914	1913	1912	1911	1910
	All causes under one year of age	110.7	102.9	105.4	103.4	108.7	116.0	112.2	116.0	131.7
5	Measles	1.1	1.1	1.4	0.7	0.7	1.5	0.9	1.0	1.3
6	Scarlet fever	0.1	0.1	0.1	0.1	0.1	0.2	0.2	0.2	0.3
7	Whooping cough	3.3	2.3	2.5	2.1	2.4	2.5	1.3	2.8	3.0
8	Diphtheria and croup	0.4	0.5	0.5	0.5	0.6	0.5	0.5	0.6	0.8
9	Influenza	6.9	0.8	1.0	0.7	0.3	0.4	0.3	0.5	0.4
(12)	Dysentery	0.3	0.2	0.2	0.3	0.3	0.3	0.4	0.4	0.6
(12)	Erysipelas	0.5	0.6	0.6	0.5	0.5	0.6	0.6	0.6	0.6
(37)	Tetanus	0.1	0.1	0.1	0.1	0.1	0.1	0.2	0.2	0.2
13	Tuberculosis of the lungs	0.5	0.6	0.6	0.5	0.6	0.6	0.6	0.7	0.8
14	Tuberculous meningitis	0.8	0.8	0.8	0.9	0.8	0.9	0.9	1.0	0.9
15	Other forms of tuberculosis	0.2	0.2	0.3	0.3	0.3	0.3	0.3	0.3	0.4
(37)	Syphilis	1.1	1.3	1.4	1.3	1.3	1.3	1.3	1.3	1.3
17	Meningitis	0.9	0.9	0.8	1.0	1.1	1.2	1.3	1.6	2.0
(37)	Convulsions	1.2	1.1	1.3	1.7	2.2	2.5	2.7	3.0	3.6
19	Organic diseases of the heart	0.4	0.4	0.4	0.4	0.4	0.6	0.6	0.7	0.8
20	Acute bronchitis	2.5	2.7	2.4	2.5	2.6	2.9	3.1	3.0	3.7
22	Pneumonia	5.6	4.7	4.2	4.5	4.8	5.3	5.7	5.6	6.9
(23)	Bronchopneumonia	10.7	9.8	9.6	10.1	9.9	9.9	9.3	8.6	8.9
24	Diseases of the stomach	1.5	1.3	1.5	1.6	1.8	2.2	2.3	2.3	2.6
25	Diarrhea and enteritis	22.2	23.2	24.1	22.6	24.7	28.1	26.2	29.0	37.7
(33)	Malformations	7.5	7.5	7.5	6.9	7.3	6.8	6.5	6.4	6.6
(33)	Premature birth	22.1	21.1	21.2	20.3	20.1	20.0	19.1	18.4	17.5
(33, 37)	Congenital debility	10.1	9.4	10.3	11.9	13.4	15.5	15.0	14.6	13.2
(37)	Injuries at birth	3.9	4.6	4.4	4.3	4.2	3.9	3.7	3.7	3.2
35	External causes	1.1	1.4	1.2	1.1	1.2	1.3	1.2	1.5	1.3
38	Unknown or ill-defined diseases	1.1	1.1	1.0	0.9	1.0	1.0	1.1	1.1	5.8
	All other causes	4.6	5.2	6.0	5.5	5.6	5.7	6.0	6.7	7.2

From U. S. Mortality Statistics, 1918, p. 47, Nineteenth Annual Report Bureau of the Census, Washington, D. C., 1920.

TABLE 6.—PER CENT. OF DEATHS FROM CERTAIN IMPORTANT CAUSES TO TOTAL DEATHS UNDER ONE YEAR OF AGE IN THE REGISTRATION STATES AS OF 1910 (EXCLUSIVE OF NORTH CAROLINA): 1910 TO 1919

Cause of death	1919	1918	1917	1916	1915	1914	1913	1912	1911	1910
All causes under one year of age	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Measles	0.5	1.0	1.0	1.4	0.7	0.7	1.3	0.8	0.9	1.0
Scarlet fever	0.1	0.1	0.1	0.1	0.1	0.1	0.2	0.1	0.2	0.2
Whooping cough	1.4	3.0	2.2	2.4	2.1	2.3	2.1	2.1	2.4	2.4
Diphtheria and croup	0.5	0.4	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.6
Influenza	4.0	6.3	0.8	0.9	0.6	0.3	0.4	0.3	0.4	0.3
Dysentery	0.2	0.2	0.2	0.2	0.2	0.3	0.3	0.3	0.3	0.5
Erysipelas	0.5	0.4	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5
Tetanus	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.2
Tuberculosis of the the lungs	0.5	0.5	0.6	0.5	0.5	0.5	0.5	0.6	0.6	0.6
Tuberculous meningitis	0.7	0.7	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.7
Other forms of tuberculosis	0.2	0.2	0.2	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Syphilis	1.1	1.0	1.3	1.3	1.3	1.2	1.2	1.1	1.2	1.0
Meningitis	0.8	0.8	0.9	0.8	0.9	1.0	1.0	1.1	1.4	1.5
Convulsions	1.1	1.1	1.1	1.2	1.6	2.0	2.1	2.4	2.6	2.7
Organic diseases of the heart	0.4	0.3	0.4	0.3	0.4	0.4	0.5	0.6	0.6	0.6
Acute bronchitis	*2.5*	*2.3*	*2.6*	*2.3*	*2.4*	*2.4*	*2.5*	*2.7*	*2.6*	*2.8*
Pneumonia	*3.7*	*5.1*	*4.6*	*4.0*	*4.4*	*4.5*	*4.5*	*5.0*	*4.8*	*5.3*
Bronchopneumonia	*9.8*	*9.6*	*9.5*	*9.1*	*9.8*	*9.1*	*8.5*	*8.3*	*7.4*	*6.8*
Diseases of the stomach	1.5	1.4	1.2	1.4	1.5	1.7	1.9	2.0	2.0	2.0
Diarrhea and enteritis	*19.4*	*20.0*	*22.5*	*22.9*	*21.9*	*22.8*	*24.2*	*23.3*	*25.0*	*28.6*
Malformations	8.0	6.8	7.3	7.1	6.6	6.7	5.9	5.8	5.5	5.0
Premature birth	*21.8*	*20.0*	*20.5*	*20.1*	*19.6*	*18.5*	*17.2*	*17.0*	*15.8*	*13.3*
Congenital debility	*9.6*	*9.1*	*9.2*	*9.8*	*11.5*	*12.3*	*13.3*	*13.4*	*12.6*	*10.0*
Injuries at birth	*4.5*	*3.5*	*4.4*	*4.2*	*4.2*	*3.9*	*3.4*	*3.3*	*3.2*	*2.4*
External causes	1.1	1.0	1.3	1.2	1.1	1.1	1.1	1.1	1.3	1.0
Unknown or ill-defined diseases	1.1	1.0	1.0	1.0	0.9	0.9	0.9	0.9	1.0	4.4
All other causes	4.8	4.2	5.1	5.7	5.4	5.2	4.9	5.4	5.8	5.5

U. S. Mortality Statistics, 1919. Twentieth Annual Report, Department of Commerce, Bureau of the Census, Washington, D. C., 1921, p. 54.

Table 7.—Per Cent. of Total Deaths under One Year of Age to Total Deaths, and in Each of the 12 Subdivisions of the First Year of Life to Deaths under One Year of Age in the Registration States* as of 1910 (Exclusive of North Carolina): 1910 to 1919

Age	1919	1918	1917	1916	1915	1914	1913	1912	1911	1910
Deaths under one year of age to total deaths...	14.4	13.0	15.7	16.1	16.6	17.3	17.9	17.7	17.9	19.4
Total deaths under one year of age.	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Under 1 day......	16.6	14.9	15.8	15.6	16.0	14.6	13.3	12.7	11.8	9.8
1 day............	5.2	4.9	4.9	5.0	5.0	5.1	4.9	5.2	5.3	4.9
2 days...........	4.0	3.6	3.8	3.8	3.7	3.8	3.4	3.5	3.3	2.9
3 to 6 days........	7.2	6.4	7.2	7.2	7.1	7.3	6.7	6.9	6.8	6.1
1 week...........	6.6	5.9	6.2	6.0	6.2	6.3	6.2	6.2	6.1	5.7
2 weeks...........	4.5	4.3	4.6	4.4	4.7	4.8	4.7	4.8	4.8	4.6
3 weeks but under 1 month........	3.6	3.4	3.7	3.5	3.7	3.7	3.7	3.9	3.7	3.8
1 month..........	8.5	8.4	8.9	8.8	9.0	9.2	9.5	9.6	9.8	9.9
2 months.........	6.8	7.0	7.0	7.0	7.3	7.6	7.7	8.0	7.9	8.4
3 to 5 months.....	16.0	16.4	16.2	16.2	16.3	16.7	17.5	17.3	17.7	19.3
6 to 8 months.....	11.9	13.6	12.0	12.4	11.9	12.0	12.5	12.4	12.8	14.1
9 to 11 months....	9.1	11.3	9.7	10.1	9.1	9.1	9.9	9.6	9.9	10.7

* *Ibid.*, Table 6.

From the English statistics, which are much more complete and cover a wider range in time, we can draw more reliable conclusions. Table 8 shows the infant mortality in England and Wales during the period 1883–1919 with the *diarrheal diseases* segregated so that we may see at once the influence of them upon the total rate. It is clearly seen that during the latter years of the last century infant mortality increased somewhat as the result of a marked increase in diarrheal diseases, while there was practically no tendency for other diseases causing infant deaths to fall. From 1900 onward there has been a precipitate fall in infant mortality, more marked since 1910. The fall in the mortality from diarrheal diseases in this period has been paralleled by a drop in mortality from other causes. The yearly fluctuations in infant mortality are often traceable to exceptionally trying summers or sudden meteorological changes.

TABLE 8.—ENGLAND AND WALES: INFANT MORTALITY, 1883–1919

Year	Deaths under one year of age per 1000 births		
	Total	Diarrheal diseases	Total less diarrheal diseases
1883	137	12	125
1884	147	21	126
1885	138	11	127
1886	149	20	129
1887	145	18	127
1888	136	11	125
1889	144	16	128
1890	151	16	135
1891	149	13	136
1892	148	15	133
1893	159	28	131
1894	137	12	125
1895	161	28	133
1896	148	21	127
1897	156	31	125
1898	160	37	123
1899	163	40	123
1900	154	28	126
1901	151	32	119
1902	133	15	118
1903	132	18	114
1904	145	30	115
1905	128	21	107
1906	132	31	101
1907	118	13	105
1908	120	20	100
1909	109	13	96
1910	105	13	92
1911	130	36	94
1912	95	8	87
1913	108	19	89
1914	105	17	88
1915	110	15	95
1916	91	11	80
1917	96	10	86
1918	97	10	87
1919	89	9	80

Eighty-second Annual Report of the Registrar-General of Births, Deaths, and Marriages in England and Wales (1919), Table XXVI.

Contributing Factors.—While the tables give some idea of the principal causes of infant mortality they do not clearly indicate any of the *contributing factors*. These may be prenatal, natal or postnatal and due to purely local conditions or more general causes. To gain some idea of the number and complexity of these conditions the most important of them are here listed with no attempt to show their relative importance or mutual relationships, intimate as these may be:

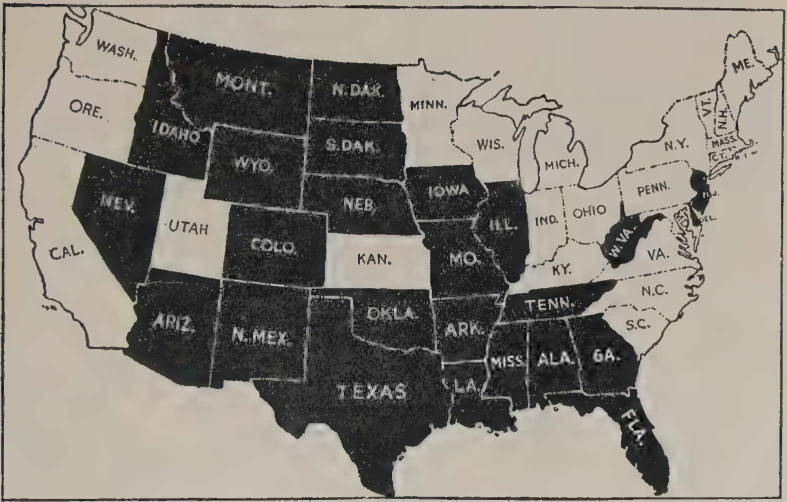
FACTORS INFLUENCING INFANT MORTALITY

- *Meteorological*
  - Variations in temperature
  - Humidity
  - Prevailing winds
  - Dust storms
  - Sunshine and fogs
- *Character of the Population*
  - Racial stamina and resistance
  - General intelligence
  - Habits and customs
  - Age distribution
  - Homogeneity
  - Diversity of language
  - Industrial welfare
  - Stability of residence
- *Births*
  - Changes in completeness of birth registration and notification
  - Marked decrease or increase in birth rate
  - Relative number of first-born in any one year
  - Proportion of male to female births
  - Proportion of legitimate to illegitimate births
  - Number of still births
  - Magnitude of birth rate
  - Attendants at birth
- *Nationality*
  - Manners and customs
  - Prevalence of breast feeding
  - Racial immunity
  - Adaptability to new environment
  - Whites *vs.* Negroes
- *Standards of Public Health and Hygiene*
  - Control of epidemics
  - Milk and water supplies
  - Character of prenatal and obstetric care
  - Housing conditions
  - Domestic and municipal sanitation.
  - Fly and mosquito prevention
  - Prevalence of vaccination against smallpox
  - Training of physicians and nurses in infant welfare
  - Provisions for treatment of syphilis and tuberculosis
  - Methods of infant feeding
  - Supervision of midwives
  - Supervision of boarding homes
- *Pathologic Conditions*
  - Prenatal pathologic states
  - Congenital defects
  - Prematurity
  - Syphilis
  - Tuberculosis
  - Alcoholism
  - Prevalence of epidemics
- *Condition of Mother*
  - Poverty and bad social life
  - Shiftlessness and ignorance
  - Industrial employment
  - Age at marriage
  - Frequency of pregnancies
  - Urban or rural life
  - Industrial poisoning
  - Malnutrition of mother
  - Exhausting diseases
- *Social and Economic Condition*
  - Unemployment. Strikes
  - Food shortage
- *Wars and Their Aftermath*

Birth Registration.—It is readily seen that so many possible factors may enter into the problem of infant mortality that no sweeping statement can be made which will adequately cover it and focus our attention upon any panacea for its reduction. Before we can gain a full rounded picture of infant mortality in the United States we must have more complete and accurate vital statistics covering a larger part of the country. A satisfactory and conclusive study of the mortalities of infancy in the United States is impossible without accurate and fairly complete statistics. Birth registration is a *sine qua non* of an exact knowledge of infant mortality and of any practical methods for its reduction, *the rate of infant mortality being most con-*

veniently expressed as the number of deaths of infants under one year of age per 1000 *born alive during the year.* Unfortunately the United States lags far behind England and Wales and other European states in the completeness of its birth returns. The registration of births, deaths and marriages in England and Wales has been remarkably complete for many years, the registration of births and deaths being made compulsory in 1870. The Annual Reports of the Registrar-General of England and Wales are admirable studies in vital statistics. The Birth Registration Area of the United States, which was established by the Director of the Census in 1915, includes those states having satisfactory registration laws which, upon tests by the Bureau of the Census, Division of Vital Statistics, can show that at least 90 per cent. of their births are registered. In 1920, 23 states and the District of Columbia were in the Registration Area for Births. This area contains 58.4 per cent. of the population of the United States

(*Reprinted from August, 1920, Number of* MOTHER AND CHILD)



BIRTH REGISTRATION AREA IN WHITE, 1920

FIG. 5.—See report of the committee on vital and social statistics on registration of births in the United States. Trans. American Child Hygiene Assoc. Annual Meeting, 1920, pp. 231–232.

(Fig. 5). The registration of deaths is much more complete, the Registration Area for Deaths in 1919 including 33 states, the District of Columbia and 18 cities in non-registration states, the total having a population of 81.1 per cent. of the total estimated population of the United States. While the certification as to the *fact* of death in this country is quite complete, the *cause* assigned to many an infant death is too often misleading. It is unfortunate that no uniform method of registering still births is in force in this country, as a considerable amount of valuable information could be gained from a careful analysis of the still births. The United States Bureau of the Census has only undertaken to compile the still birth statistics for the one year 1918. For a number of years the New York Milk Committee collected statistics on infant mortality and made them available each year before it was possible for the Census Bureau to compile its data. In 1912 the Committee issued a special report which contains a wealth of statistical data and other valuable material.* In this way excellent educational work was carried on which stimulated many communities to organize their resources for the reduction of infant mortality. During the past two years the American Child Hygiene Association has continued the statistical reports on infant mortality of the New York Milk Committee by collecting statistics on infant mortality in the United States and making them available as early as possible. In 1920 the American Child Hygiene Association published statistics covering 269 cities of over 10,000 population in the United States for 1919. In June, 1921, a statistical report of infant mortality for 1920 in 519 cities in the United States was published.† When complete and prompt birth notification and registration are carried on in all parts of the country we may expect to have decidedly lower infant mortality rates. We will then be in a position to trace from year to year precisely what is taking place in the mortalities of infancy.

The Trend of Infant Mortality in the United States.—The trend of infant mortality for the 519 cities included in the 1921 Report of the American Child Hygiene Association is shown according to population grouping in Table 9.

The figures for the entire 519 cities for the five years follow:

TABLE 9.—INFANT MORTALITY RATES

POPULATION	1916	1917	1918	1919	1920
Over 250,000	97.9	95.0	102.9	86.9	88.9
100,000–250,000	107.0	104.0	116.1	92.8	94.1
50,000–100,000	103.0	100.3	105.5	89.7	93.9
25,000–50,000	103.1	95.2	106.3	88.2	87.5
10,000–25,000	105.3	100.7	113.0	93.9	91.6
All cities in Area	101.1	98.1	106.8	89.0	90.5

It is seen that a distinct gain in the saving of infant life has been made even in the last five years, and this is more marked if we carry our observations back over a 10-year period. One of the most interesting and hopeful signs is that the smaller cities are showing each year a relatively greater reduction than the larger centers of population. This indicates on the whole that the smaller towns are beginning to learn the lessons which were being taught in the larger cities during the preceding years. This "catching up" is well shown in Fig. 6

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* Infant Mortality and Milk Stations. Special Report of the New York Milk Committee on the Reduction of Infant Mortality, edited by Philip Van Ingen and Paul Emmons Taylor. New York City (1912).

† Statistical Report of Infant Mortality for 1920 in 519 Cities of the United States. Compiled by Philip Van Ingen for the American Child Hygiene Association, 1211 Cathedral Street, Baltimore, Md. (1921).

giving the infant mortality rates monthly from 1913 to 1919 for New York City, New York State and for parts of the state outside New

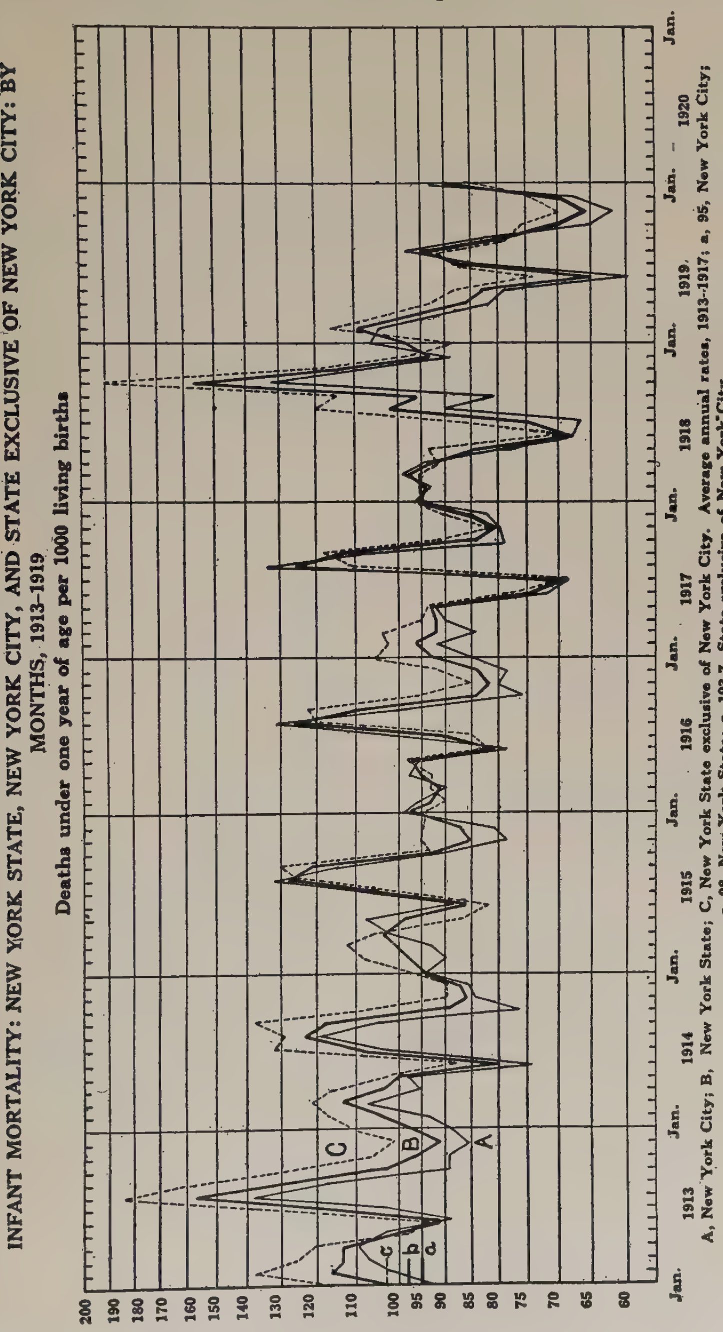


FIG. 6.—From the monthly vital statistics review of the New York State Department of Health for March, 1920. Otto R. Eichel, statistician.

York City. A comparison of what has been accomplished in Utica with New York City is also instructive (Figs. 7, 8 and 9).

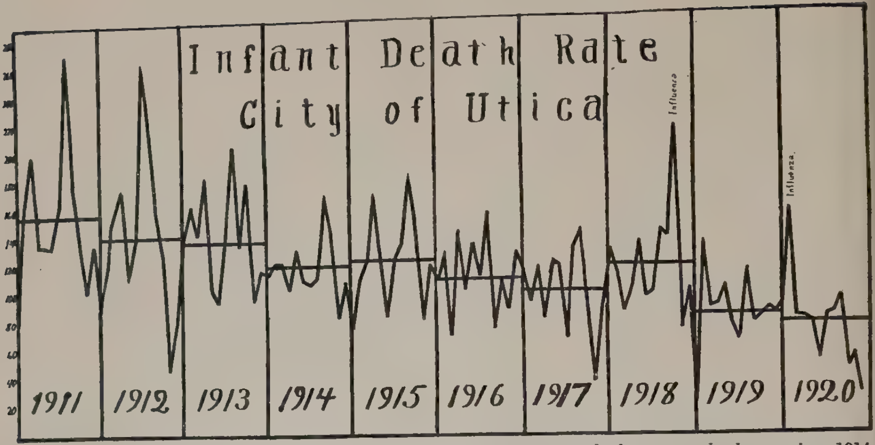
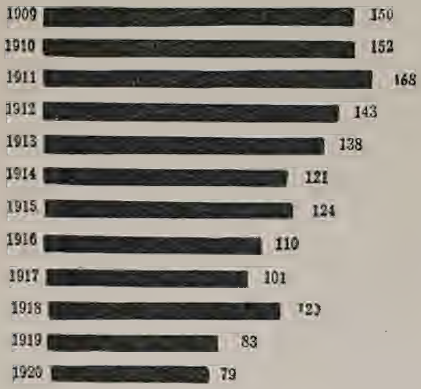


Chart showing infant death rate per month in Utica. Note disappearance of high summer death rate since 1914. *Compare death rates for influenza months of* 1918 *and* 1920 *with ordinary death rates in* 1911 *and* 1912.

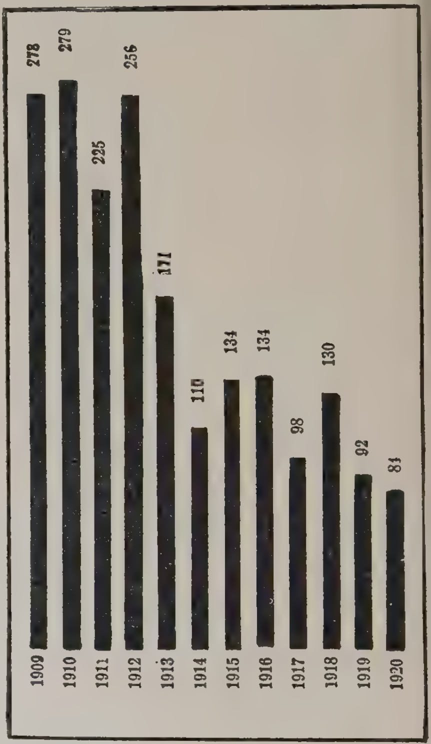
Fig. 7.



Infant Mortality In Utica.

Note rise in infant death rate prior to organization of Utica's Baby Welfare Committee in 1912 and steady decline since that year, except in 1918, the year of the influenza epidemic.

Fig. 8.



Summer Infant Death Rate In Utica.

Deaths per 1,000 births for months of July and August.

Fig. 9.

Figs. 7, 8 and 9.—Cutting Down Death Toll Among Utica's Babies by T. Wood Clark, M.D., Mother and Child, Vol. 2, No. 10, pp. 458–463 (October, 1921).

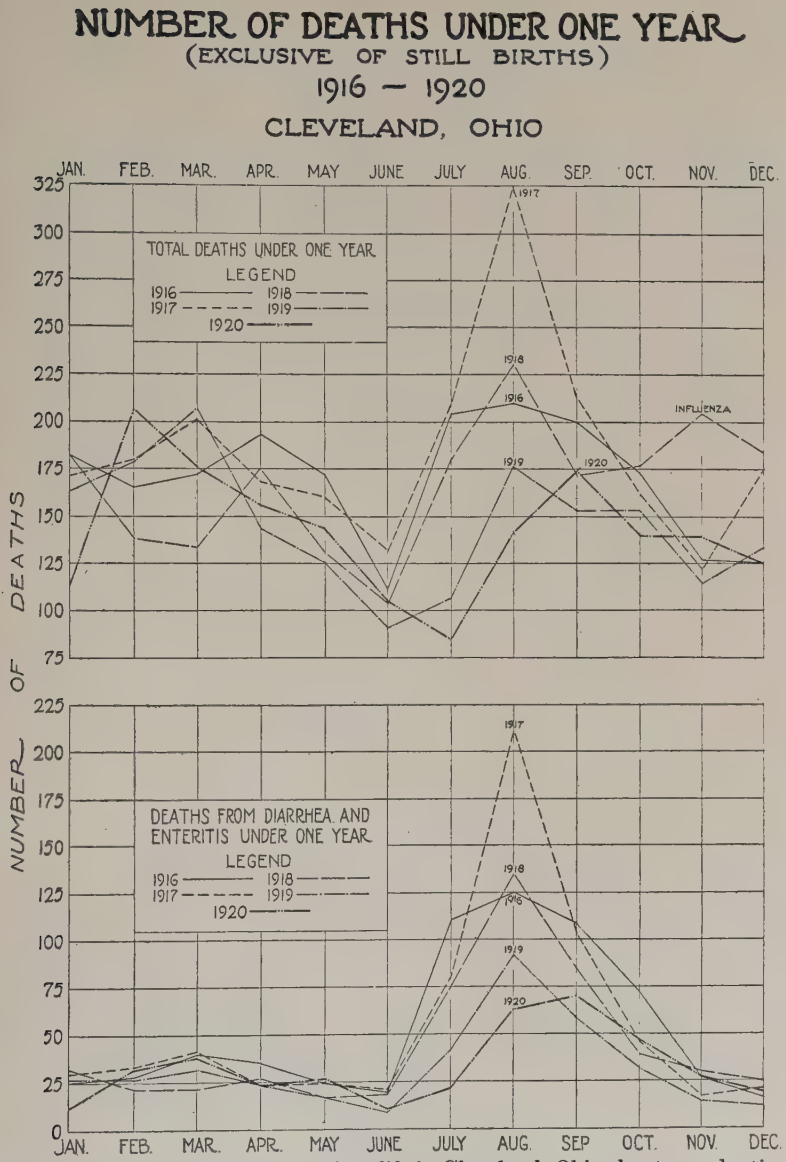


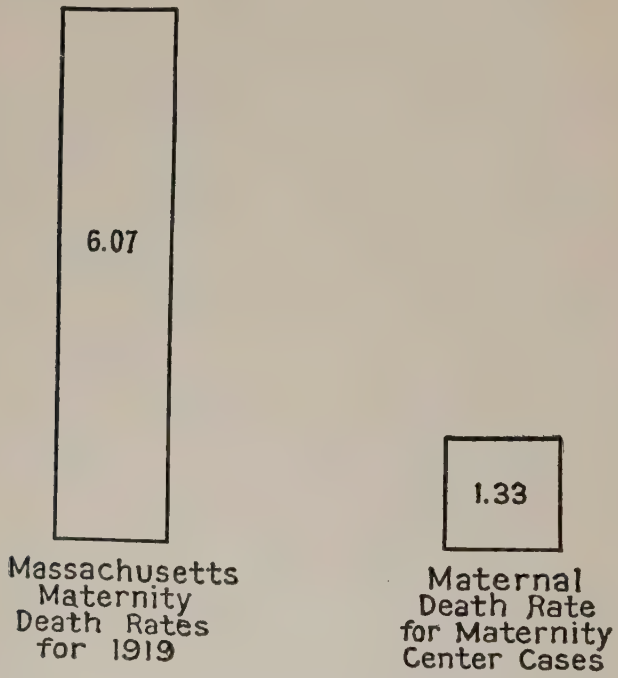
Fig. 10.—Showing the saving of infant life in Cleveland, Ohio, due to a reduction in gastro-intestinal deaths.

(From Data Supplied by Dr. R. J. Ochsner of Cleveland.)

What then has been our actual gain in infant life during the past decade in the United States? In 1900 the deaths under one year of age made up 20.7 per cent. of the total number of deaths at all ages. In 1910 it was 19.4 per cent. and in 1919 deaths under one year of age comprised only 14.4 per cent. of the total deaths. Stated in another way, in 1900 of every eight infants born alive approximately one died before reaching its first birthday, while in 1919 of every 12 born alive only one died before the age of one year. This saving of life is due largely to great reduction in deaths from gastro-intestinal diseases, and in minor degree to a reduction in acute infectious diseases. Table 5 shows the great reduction which has taken place in gastro-intestinal diseases as compared to other diseases of childhood. Taking the city of Cleveland, Ohio, as a typical large industrial community we see in plotting the actual number of infant deaths by months each year for the five-year period 1916–1920 that there has been a remarkable reduction in the total infant deaths. On the same graph will be noted the number

RESULTS OF WORK OF THE MATERNITY CENTER ASSOCIATION OF NEW YORK

Of 4,500 cases cared for by the association in 1919, there were 6 maternal deaths from causes due to pregnancy or childbirth.



NEW YORK CITY

FIG. 11.—Showing that the maternal death-rate can be considerably lowered by suitable prenatal care.

of deaths from diarrhea and enteritis under one year of age for the corresponding period. The decline in total infant deaths is evidently due in large measure to the reduction of deaths from gastro-intestinal diseases (Fig. 10).

Another very interesting point in regard to this graph is that the summer peak of infant deaths, which parallels the peak for diarrhea and enteritis, has been coming down year by year until now the winter peak is higher than the summer. This indicates, in a striking way, the necessity for more concerted attack upon the respiratory diseases which now predominate during the winter months. A closer analysis of Cleveland's figures under one year of age revealed a very large proportion of deaths under one month and a high still-birth rate. Knowing, as we do, that the number of still births and deaths under one month can be considerably reduced by suitable prenatal and obstetrical care (see Figs. 11 and 12 which illustrate what has been done in New York and Boston) there is every reason to believe that the infant mortality rate can be still further reduced not only in Cleveland but in every other American community. For instance, in a small well-to-do community such as Montclair, New Jersey, the infant mortality rate in 1920 was only 58, but 46.6 per cent. of the deaths under one year occurred under one week of age.

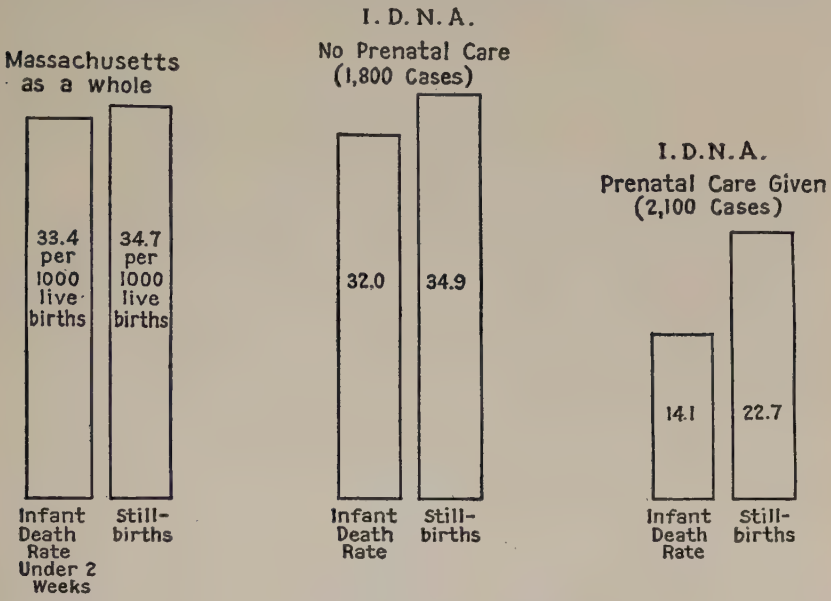


FIG. 12.—Showing reduction of still births and early infant deaths in cases given prenatal care by the Instructive District Nursing Association of Boston.

Prenatal and Neonatal Mortality.—To the layman birth is looked upon as the most important event in life. It signifies the completion of Nature's fruition and marks the beginning of a new life. We know, however, that birth is by no means the beginning of life, but simply an incident, fraught with many dangers and possibilities, in the larger life of the community. No study of infant mortality, therefore, is complete until we have looked back into prenatal life and considered the possibilities of the potential loss and damage which may not be revealed until after birth.

The study of prenatal life is beset with many difficulties, but an excellent beginning has been made by Ballantyne, Feldman and others. The study of infant mortality should really begin with ante-natal pathology and hygiene. The problems of ante-natal and neonatal mortality have a very intimate relation to the welfare of the mother. The environment of the fetus and newborn is the mother, and any pathologic conditions which affect her react unfavorably upon the infant. It should be borne in mind that the fetus exhibits varying reactions to its environment at different periods of its growth. This has been clearly pointed out by Ballantyne. The high still-birth rate and neonatal mortality reflect seriously upon our care of the pregnant and parturient woman. The high rate of maternal mortality in this country undoubtedly has a direct influence on the mortalities of early infancy. Ballantyne has so well stated the loss in prenatal and neonatal life that his statement is given here in full. "If one takes infantile mortality as 100 per 1000 live births (and that rate is still far exceeded in many places) then something between a half and a third of all these infantile deaths are found to occur in the first month of life. Between 40 and 45 of the infantile deaths are neonatal (i.e., occur in the first month after birth). Obviously, if the fatalities are evenly distributed over the 12 months the neonatal ones should be only 8 or 9; instead of this, more than 40 of them are closely packed into the first month, and if this number were continued during the remaining 11 months the infantile deaths would number 500 out of every 1000 live births.

"The neonatal deaths are serious enough, but the ante-natal ones are far more serious. It will have been noticed that in estimating the infantile and neonatal deaths one always speaks of them as 'so many in 1000 *live* births;' that is to say, they are a superincumbent mortality, and already other deaths have occurred in birth and before birth of which no record is preserved in the 1000. In other words, the dead births require to be added, and there are some 30 or 40 of them in each 1000 births. But even this is not all: every miscarriage means an ante-natal death, for the unborn child is not viable in a miscarriage. There is no certain knowledge regarding the miscarriage rate, but it may be safely stated to be not less 150 per 1000 conceptions. Putting all these facts together the conclusion is reached that if one starts with 1200 conceptions something like 200 of these lives are lost before pregnancy is ended and labor completed; and of the 1000 infants who come into the world alive 40 or 45 die in the first month, i.e., neonatally. Consequently a completely successful campaign to abolish all ante-natal and neonatal deaths would mean the saving of 230 or 240 lives in each 1200 conceptions, or to state it in the milleage, 200 per 1000. Obviously there is a wide field for life-saving endeavors among the ante-natal and neonatal deaths."[*]

Miscarriages and abortions make up a very large proportion of all pregnancies. Although it is quite difficult to obtain exact statistics regarding abortion it is known from the data we do possess that it is quite frequent. According to a number of obstetricians *one out of every five pregnancies ends in abortion.* Williams admits the difficulty of arriving at any very accurate conclusions regarding abortions, especially in this country. He states that in his maternity service at Johns Hopkins Hospital about 6 per cent of all his patients suffer from abortion.[†] Referring to Mallins he gives 19.23 per cent of the pregnancies of 2,000 patients ending in abortion, and to Franz that in 15.4 per cent. of the cases admitted to the Lying-in Hospital at Halle abortion occurred.[‡]

It is conservatively estimated that in private practice about one-fifth or one-sixth end in abortion, although Taussig thinks that one abortion occurs to every 2.3 labors and he estimates that over 25 per cent. of all abortions are "criminally induced."[§] The seriousness of induced abortions has received particular attention in Europe for some time. In France where "Birth Control" has been the vogue, abortions have increased at such an alarming rate as to cause the statesmen great anxiety. It was found by Doleris that in the poorer arrondissements of Paris the number of abortions had doubled in some, and even trebled in others, in the years between 1897 and 1905. G. Rimette[‖] compiling the statistics of the Paris Maternité from 1897 to 1905 found:

9875 pregnancies
1437 abortions
627 spontaneous abortions
414 complicated abortions
367 infected abortions
27 deaths from abortion.

Miscarriages and Still Births.—When we come to the miscarriages and still births we still find very high rates. Whitehead found the proportion of miscarriages to pregnancies as 1:7 and Priestly as 1 : 4⅓.[¶] For the European countries Wappaus estimated the still births as 3.79 per cent. of the total births and Rippin gives the still births in Prussia for 25 years as 3.69 per cent. of the total live births.* In this country we have no reliable statistics of still births covering a large area. The figures given for the 1918 Census Bureau Report were 3.9 per 100 live births for legitimate and 9 per 100 illegitimate births.† Where figures are given we know that they are considerably below the actual number of still births, for a large number of them are never reported. It is estimated on the figures we have that to every 100 live births there are between four and five still births. In New York City there have been reported as many as six still births to every 100 live births in a single year. In the state of Massachusetts for the year 1919, 3046 still births were reported, which makes a rate of 34.7 per 1000 reported live births. An inquiry sent out by the Metropolitan Life Insurance Company in 1919 to the health officers of all states and larger cities having good birth registration revealed that:

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[*] Ballantyne, J. W., The Prevention of Ante-natal and Neonatal Mortality, Nelson Loose Leaf Medicine, vii. Prevention of Disease—Public Health, Chapter XVIII, pp. 436–437. Thos. Nelson and Sons, N. Y. and London (1920).

[†] Williams, J. Whitridge, Obstetrics, Fourth Edition. D. Appleton and Co., N. Y. (1917), p. 661.

[‡] *Ibid.*, p. 661.

[§] Taussig, Frederick J., The Prevention and Treatment of Abortion, pp. 4–5. C. V. Mosby and Co., St. Louis (1910).

[‖] *Ibid.*, p. 3.

[¶] Ballantyne, J. W., Disease of the Fetus, i (1892), p. 8.

"1. More than five women die from disorders of pregnancy or childbirth out of each 1000 births registered. This is equivalent to one maternal death out of every 185 confinements.

"2. Forty-five babies out of every 1000 total births, or one out of every 22 are born dead.

"3. Forty babies out of every 1000 born alive die before they are one month old."‡

Birth Control.—This terrific loss of life *in utero* or shortly after birth constitutes a serious problem from a biological as well as a social standpoint. Of recent years there has been an alarming increase in the frequency and actual number of induced abortions. This has gone hand in hand with the insidious propaganda for so-called "birth control" or "voluntary parenthood"—a movement which has gained momentum in France, Holland and New Zealand, and has gradually spread to England and the United States. While the birth control enthusiasts would indignantly disclaim any connection between the "contraceptive methods" of "voluntary parenthood" and induced abortions, it is very evident that the more "moral technique" of contraception must often break down and relief from the "accidents" which follow be frequently sought in induced abortion. Thus far contraceptive methods have been practiced largely by the elite and better educated classes. Those most able to bear children and meet their support have been the very ones to shirk the responsibility, while those for whom birth control is claimed to be a great boon still proceed to build up large families. It has been estimated that at least four children to a family are necessary to keep up the stock.

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* Cited by Griffith, J. P. Crozer, The Diseases of Infants and Children, i, (1919), p. 211. W. B. Saunders Co., Philadelphia (1919). Allg. Bevölkerungsstatistik, 1859. Ref. Pfeiffer, Gerhardt's Handb. d. Kinderkr., i, p. 551. Also Schmidt's Jahrbücher, cclxxiii (1902), 233.

† Birth Statistics for the Birth Registration Area of the U. S., 1918, p. 30. Fourth Annual Report. Bureau of the Census, Washington, D. C. (1920).

‡ Statistical Bulletin, Metropolitan Life Ins. Co., Louis I. Dublin, Statistician i, No. 3 (March, 1920), p. 1.

This standard is not even being maintained in the United States. There is no question but that the native American stock is rapidly dying out and is being replaced by those races or mixture of races which do *not* refuse to bear children. Note the Russian and Polish Jews on the Atlantic Coast and the Italians and Japanese on the Pacific slope. "Birth control" is simply a refined twentieth century paganism with regard to the undesired child which seeks in the guise of humanity and the argument of "economic necessity" to obtain the maximum of sexual stimulation without incurring the difficulties and responsibilities of parenthood. The only "birth control" that can be considered really "moral" is strict continence, which in theory, at least, has been held by the Jewish and Roman Catholic churches for centuries as a protest against "pagan practices."

The Declining Birth Rate.—It is instructive to recall that a study of the declining birth rate in France was what first led to an intensive investigation of the causes of infant mortality and means for their prevention. The decline of births in France after 1870 led to a condition rapidly approaching national disaster when the deaths threatened to exceed births. A Commission on Depopulation was appointed to make a thorough study of the whole question. The principles evolved from the French study cannot be better stated than as given by Sherman C. Kingsley who paraphrases the Report of the Commission on Depopulation in 1909 giving reasons for the rapid decrease in the number of births (several other causes have been added by the writer):

"1. The spread of the Neo-Malthusian doctrines, which justified the prevention of conception.

"2. The increase of venereal disease, which rendered marriages sterile (or resulted in repeated abortions).

"3. Industrial conditions, which imposed upon working classes poor food, insanitary dwellings and increased employment of women in factories and shops.

"4. The law which forbade investigation of the parentage of an illegitimate child.

"5. The increased ease with which divorce could be obtained.

"6. The lack of special laws for the protection of pregnant women."*

7. (The laws of inheritance which favored the first born and made little provision for the succeeding children in the inheritance of the property.)

While the decrease in the number of children born undoubtedly overshadowed the danger of deaths through the first year of life, measures to prevent infant mortality formed an important part of the work of the Commission on Depopulation. Among these measures may be included:

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* Kingsley, Sherman C., "Infant Welfare Activities in European Countries." See "Steps in the Evolution of Baby Welfare Work in Chicago," Pub. by Elizabeth McCormick Memorial Fund, 1914. "Significant Measures in Germany," pp. 25–27.

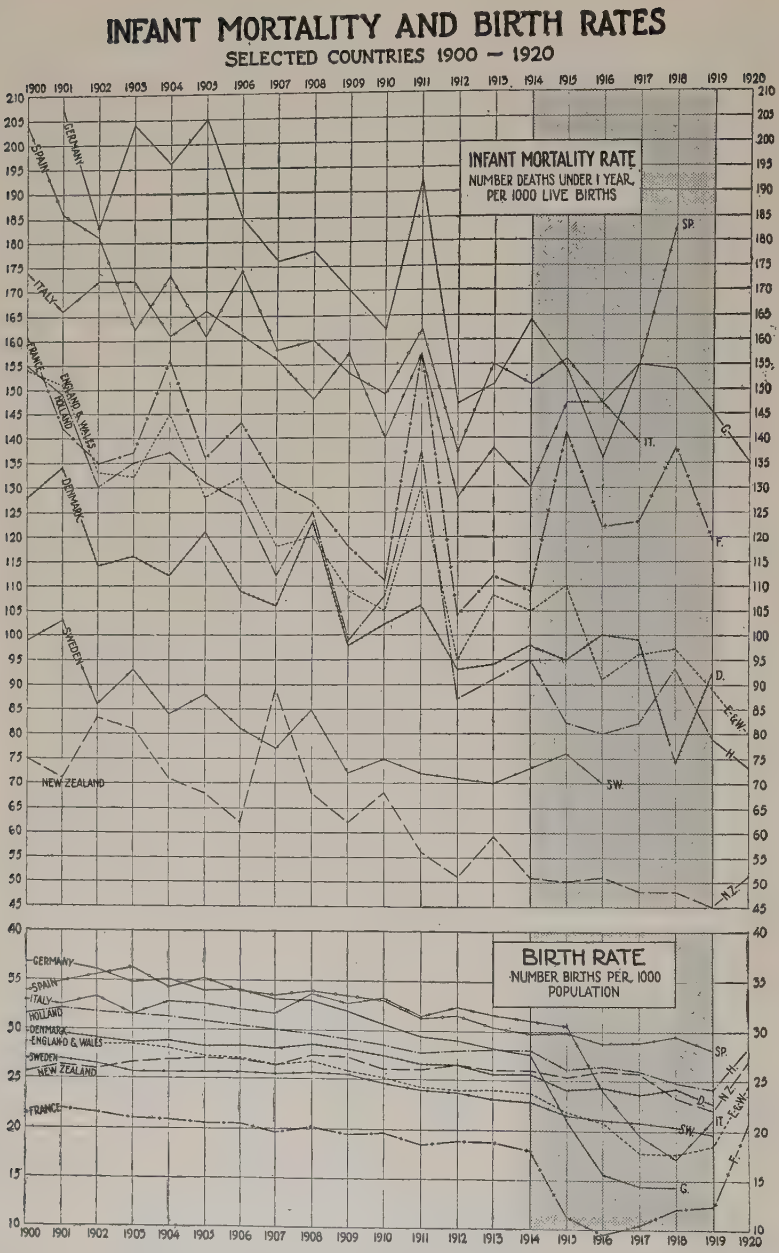

Fig. 13.—The effects of the Great War upon the birth rates and infant mortality are shown in the shaded sections.

1. Consultations for pregnant women.
2. Refuge homes for unmarried mothers and working women.
3. Money grants during pregnancy.
4. Maternity hospitals.
5. Care of women in home during confinement.
6. "Canteens" or restaurants in the poorer quarters of Paris, where any nursing mother could get a meal for a nominal amount, or free of charge if she was too poor to pay. These efforts in France were coupled with the establishment of consultations for nurslings, "gouttes des lait," crèches, etc., which give to France the distinction of being the pioneer in modern organized maternity and child hygiene work.

The declining birth rate in other countries soon attracted the attention of statisticians and statesmen. Efforts were put forth with the hopes of checking it, but with little if any success. It was reasoned that if little could be done to check the decline, much might be done to prevent infant mortality among the smaller number of children born, and hence, this became a stimulus to infant welfare, notably in Germany and Austria. In these latter countries maternity benefits and the promotion of breast feeding were soon important features.

Birth Rates and Infant Mortality Compared.—It is instructive to compare the decline in the birth rates with the fall in infant mortality. This can be readily seen in Fig. 13 where the birth rates and infant mortality rates are given for some of the European countries, over a series of years. The war years have been included so that the bearing of this catastrophe upon infant mortality could be studied. While a high birth rate is often accompanied by a high infant mortality, this is not invariably the case.* In Ireland, for instance, where the birth rate is relatively high, the infant mortality rate is surprisingly low. Since the war the birth rate in England has steadily increased while the infant mortality rate has continued to fall. Conversely a low infant mortality rate is not guaranteed by a low birth rate. It has been noted during certain wars and in other national disasters that while the births dropped off considerably there was not a corresponding increase in infant deaths, e.g., in France during the Siege of Paris in 1870, and during the Lancashire cotton famine, and in England during the late war specific measures for the reduction of infant mortality appear to have more of an influence upon it than fluctuations in the birth rate. There are, however, certain corrections to be made when the number of births falls abnormally low in any one year, and this has been clearly pointed out by the Registrar-General† of England regarding the infant mortality rates in that country during the war.

Factors Influencing the Infant Mortality Rate.—The proportion of male to female infants does have a distinct bearing upon the infant mortality, as the deaths of male infants are uniformly higher than those of female (Table 4). This is also seen in the table of infant mortality according to sex given for Germany for a series of years (Table 10).

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* Newsholme, Sir Arthur, Address on Neonatal Mortality, Mother and Child, i, No. 1 (June, 1920), pp. 3–4.

† See Eightieth Annual Report of the Registrar-General of England and Wales for 1917, p. 29.

TABLE 10.—INFANT MORTALITY IN GERMANY BY SEX, 1901–1918

Rates per 1000 living births of each sex

Years	Males	Females	Total
1901	223	190	207
1902	199	166	183
1903	220	187	204
1904	212	180	196
1905	222	188	205
1906	201	169	185
1907	191	160	176
1908	194	162	178
1909	184	154	170
1910	176	147	162
1911	207	177	192
1912	160	134	147
1913	164	137	151
1914	177	149	164
1915	167	140	154
1916	147	124	136
1917*	167	141	155
1918*	167	140	154

* Excluding Alsace-Lorraine.

Source: 1901–1916 Stat. Jahrbuch. für des Deutsche Reich. 1917–1918 Delbrück, Stat. Reichsamt, Berlin.

While the proportion of male infants has apparently increased in most countries since the War it has had but little effect in increasing the infant mortality in those countries where systematic infant welfare work has been encouraged. This illustrates an important point in infant welfare work, *viz.*, there are so many factors entering into it that one or more favorable factors may offset or balance the untoward ones. For instance, good economic conditions may do much to offset certain factors which are otherwise unfavorable, and an intensive campaign for breast feeding, or pasteurized milk, may neutralize a poor general milk supply.

The age and social status of the mother has something to do with the infant mortality rate. This has been well shown by the admirable studies of the Children's Bureau in a number of typical American cities. The infant mortality rate for infants of mothers between 25 and 29 was the lowest of any age group. It is interesting to note that the largest number of births was among this age group, which again illustrates that a high birth rate among certain age groups does not necessarily imply a high infant mortality (see Table 11).

TABLE 11.—BIRTHS DURING SELECTED YEAR, INFANT DEATHS, INFANT MORTALITY RATE, AND PER CENT. OF STILL BIRTHS, ACCORDING TO AGE OF MOTHER AT BIRTH OF INFANT

New Bedford, Mass.

Age of mother	Total births	Live births	Infant deaths	Infant mortality rate	Still births	
					Number	Per cent. of total births
All mothers	2662	2587	337	130.3	75	2.8
Under 20	112	108	28	259.3	4	3.6
20 to 24	737	725	93	128.3	12	1.6
25 to 29	853	833	95	114.0	20	2.3
30 to 39	840	809	105	129.8	31	3.7
40 and over	120	112	16	142.9	8	6.7

From Infant Mortality Study of New Bedford, Mass., by J. S. Whitney. Infant Mortality Series No. 10, Bureau Pub. No. 68, U. S. Children's Bureau, Washington, D. C., 1920.

The relative number of first-born children in any one year has a distinct bearing not only upon the number of infant deaths but upon the maternal mortality as well. On the average the first-born children have a 33 per cent. higher mortality than the second born. The still-birth rates for males and also for first-born infants are in excess of those for females and for second and following births.

The nationality and nativity of the mothers and the length of residence in this country influence to a considerable degree the infant mortality rate. Furthermore, colored populations residing in this country for some time exhibit marked differences in infant mortality. The Negro race has always had a high infant mortality compared with the white and with other colored races. Some idea of the differences in mortality can be obtained from Table 12. The differences in the mortalities of infancy among foreign born mothers was well brought out in the studies of the Children's Bureau. In small countries where nationality is more homogeneous this factor in the infant mortality is largely eliminated. The relative amount of breast feeding among the various nationalities in this country also has a definite bearing upon the infant mortality rate.

Urban and rural infant mortality rates differ considerably throughout the country, but it is difficult to make any statement which will cover broadly the influence of rural or urban life upon infants. The local conditions will largely determine the trend of infant mortality. Some idea of the variation of urban and rural infant mortality rates in the United States may be obtained from Table 13. For a detailed analysis of the causes of infant deaths in city and country the reader is referred to the Infant Mortality section of the Birth Statistics

Reports for the Registration Area of the U. S. issued by the Bureau of the Census.

TABLE 12.—INFANT MORTALITY RATES FOR THE REGISTRATION AREA OF U. S.

	1915	1916	1917	1918	1919
The registration area (total)	100	101	94	101	87
White	99	99	91	97	83
Colored	181	185	151	161	131
Cities in registration area (total)	103	104	100	108	89
White	102	102	96	105	86
Colored	181	177	185	197	148
Rural parts of registration area (total)	94	97	88	94	84
White	94	95	84	90	80
Colored	182	203	134	143	123

TABLE 13.—URBAN AND RURAL INFANT MORTALITY RATES BY STATES

Birth registration states, 1919*	Infant mortality rates		
	Total	Urban	Rural
Total	87	89	84
California	70	64	79
Connecticut	86	86	87
Indiana	79	88	74
Kansas	70	88	65
Kentucky	82	105	78
Maine	91	89	91
Maryland	105	98	115
Massachusetts	88	90	82
Michigan	90	97	82
Minnesota	67	68	66
New Hampshire	93	101	85
New York	84	85	77
North Carolina	84	124	82
Ohio	90	94	85
Oregon	63	69	59
Pennsylvania	100	99	101
South Carolina	113	139	111
Utah	71	74	69
Vermont	85	121	79
Virginia	91	106	87
Washington	63	59	67
Wisconsin	80	94	71
District of Columbia	85	85	

Source: Birth Statistics, 1919. U. S. Bureau of Census.

* N.B.—The birth registration area comprises 58.6 per cent. of the population of this country, or 61,474,111, including Rhode Island (for 1919).

Illegitimacy.—In all countries illegitimate babies have a slimmer chance of surviving the first year than the legitimate ones. In foreign countries where careful statistics are kept regarding this condition the infant mortality rate among the illegitimate is about twice as high as among the legitimate babies (Tables 14 and 15 for England and Wales, and Germany). The causes underlying this difference are not difficult to discover. Many of the illegitimate babies are premature or suffer from syphilitic infection. The social stigma which still unfortunately falls upon the mother alone prompts her early to get rid of the baby in some way. If placed in an institution its chances of surviving are further reduced, as it not only loses the personal care of its mother but also the breast milk which may be its salvation. In foundling institutions of Europe it is often possible to secure wet nurses for the unfortunate babies and thus prolong their lives. If the mother can be persuaded to keep her baby during the perilous first few months it may do fairly well, but the temptation to get out to work and leave the baby in a boarding home, or in the care of a questionable helper, is so great that the mother often capitulates. The whole problem of illegitimacy has received considerable attention during the War and since. As a cause of infant mortality aside from its social complex it deserves special attention.

TABLE 14.—INFANT MORTALITY IN ENGLAND AND WALES BY LEGITIMACY
Deaths of infants under one year per 1000 live births

Years	Legitimate	Illegitimate	Total
1906	127	261	132
1907	113	220	118
1908	116	233	120
1909	104	211	109
1910	102	195	105
1911	125	245	130
1912	91	181	95
1913	104	213	108
1914	100	207	105
1915	105	203	110
1916	87	183	91
1917	90	201	96
1918	91	163	97
1919	84	187	89

Source: Eighty-second Annual Report of the Registrar-General of Births, Deaths and Marriages in England and Wales, 1919.

Table 15.—Infant Mortality in Germany by Legitimacy
Rates per 1000 living births

Years	Legitimate	Illegitimate	Total
1901	194	339	207
1902	173	293	183
1903	193	327	204
1904	186	314	196
1905	194	326	205
1906	175	294	185
1907	166	280	176
1908	168	285	178
1909	160	268	170
1910	152	257	162
1911	182	299	192
1912	139	232	147
1913	142	237	151
1914	154	253	164
1915	144	253	154
1916	126	213	136
1917*	143	257	155
1918*	141	239	154

* Excluding Alsace-Lorraine.

Source: Delbrück, Stat. Reichsamt., Berlin, Nov., 1919.

Infant Feeding.—There is no question that the character of feeding during the first year of life is the main factor in the health destiny of the baby. Although prenatal and natal conditions may largely account for neonatal deaths we must look to difficulties in feeding as the inderlying cause of the disorders which result in so many deaths during the later months. The early establishment of breast feeding is of paramount importance. Upon this point there should be no controversy between the obstetrician and pediatrist. Valuable time is sometimes lost in the early days of life because the obstetrician in his solicitude for the mother overlooks the importance of early establishment of proper nursing habits. In every country there is unanimity of opinion as to the supreme value of breast feeding as a preventive of infant mortality. Our whole clinical experience, strongly supported by laboratory research, affirms that mother's milk is the safest and best food for the infant and by all odds the most economical. There is no real substitute for mother's milk.

It is unnecessary here to quote exhaustive figures to prove what we all know so well, that the breast fed baby's chances of surviving the first year are much greater than those fed upon proprietary foods or even modified cow's milk. While great strides have been made in the production of certified milk, in the commercial pasteurization of market milk and in the preparation of dried milks which have on the whole proved satisfactory, it still remains true that mother's milk is the milk *par excellence* and should be guaranteed to every baby when physically possible.

A decade or so ago when refinements in artificial feeding were being introduced—when the percentage method was competing with simple dilutions—there was a tendency on the part of a number of physicians to acquiesce in the wishes of their patients to wean the baby on the slightest pretext and attempt to substitute a cow's milk modification or a proprietary food. This was greatly encouraged by adroit advertising by the manufacturers of patent foods. It looked for a time as though breast feeding was rapidly going out of vogue, at least among the native born Americans. Thanks to the persistent efforts of our pediatrists and the educational work carried on by child hygiene workers, breast feeding has gradually regained its ground until in some communities from 80 to 90 per cent. of the mothers are nursing their babies. It has been shown by Sedgewick, Herman Schwartz and others* that by suitable methods of manual expression it is possible to reestablish breast feeding after long periods of apparent dryness and that it is feasible to get practically every mother to nurse her baby for at least part of the time. Hoobler† in Detroit has demonstrated the practicability of conducting a "human dairy," and of supplying human milk to mothers who could not really nurse their own babies.

Breast Feeding and Immunity.—Investigations have proved that mother's milk is not only the best protection against the gastrointestinal diseases, but also that babies taking it thrive more normally and appear to exhibit a greater immunity to the infectious diseases. If any of the infectious diseases are contracted the breast fed babies have a better chance of recovery and exhibit fewer unfavorable sequelæ. While rickets, scurvy and beri-beri have been known to develop in breast fed babies, their occurrence is relatively rare where the dietary of the mother contains the necessary food elements, and where the babies are not nursed exclusively at the breast for too long a period. Rickets and scurvy are extremely rare in China where breast feeding is well nigh universal.‡ They were practically unknown, even in the treaty ports, until artificial feeding was introduced by the foreigners. In the Orient babies fed at the breast of beri-beric mothers have developed acute beri-beri. In Manila and Japan at one time infantile beri-beri was an important cause of death.*

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* Annual Proceedings of the American Association for Study and Prevention of Infant Mortality (American Child Hygiene Association).

1. Sedgewick, J. P., Maternal Feeding, 3d Annual Meeting (1912), pp. 195–205.
2. Schwartz, Herman, Nursing Statistics Derived from the Study of the Infancy of 1500 Children and a Contribution to the Cause of the Summer Infant Mortality, 1st Annual Meeting (1910), pp. 207–215.
3. Rudd, Nathalie C. Breast Feeding Propaganda as Applied to a Birth Registration of 9000 (in Minneapolis), 8th Annual Meeting (1920), pp. 89–96.

† Hoobler, B. Raymond, Problems Connected with the Collection and Production of Human Milk, Jour. A. M. A., lxix, No. 6 (Aug. 11, 1917), pp. 421–429.

Hutzel, Eleonore L., The Detroit Bureau of Wet-nurses. Mother and Child, ii, No. 7 (July, 1921), pp. 316–319.

‡ Bolt, R. A., The Chinese Child, *ibid.*, ii, No. 3 (March, 1921), pp. 99–106.

Meteorological Factors.—It is well known that climate has an influence upon infant mortality, but just how the meteorological factors balance one another is not so well defined. The relation of excessively hot and dry summers to infant mortality has been pointed out in a number of studies. In comparing the infant mortality of different countries, or of parts of the same country, the differences in climate must always be carefully considered. For instance, the climate of New Zealand is very favorable to low infant mortality. The favorable climate is quite apt to be accompanied by more favorable housing conditions, as is also found in New Zealand. Another notable example is seen on the Pacific slope of United States where the infant mortality rates have been on the whole the lowest in this country. There is no question that cooler summers, especially when accompanied by cool nights, and milder winters have had a good deal to do with keeping the infant mortality rates down in California. It should be noted, however, that the infant mortality in the early months of life here and there throughout California is as high as in many other parts of the country. It must be remembered that in some sections of the west —in Washington and Oregon—the age distribution of the population is more favorable to a low infant mortality. It is a matter of considerable interest that while climate favors the far west, a number of cities in the middle west and the east are now showing infant mortality rates comparable to those in the west. It is fair to conclude that even the unfavorable effects of the eastern climate may be neutralized to some degree by intensive child hygiene efforts. At any rate a reduction of neonatal deaths is open to east and west alike, as climate probably has but little influence upon the early deaths.

Heat and Infant Mortality.—Several interesting studies of the relation of heat to infant mortality have been made in this country and important contributions to our knowledge have come from abroad. A most exhaustive and painstaking investigation was carried out by Liefmann and Lindermann† who studied this question for a period extending over 15 years in the city of Berlin. Figures 14 and 15 based upon their observations were prepared by Schereschewsky of the Public Health Service and two of them are presented here. Schereschewsky‡ has given an excellent summary of our knowledge regarding the relation of heat to infant mortality with an extensive bibliography up to the year 1913. He pointed out that "by far the most conspicuous phenomenon in connection with the mortality of infants is the well-

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* Vedder, E. B., "Beri-beri" (1913), pp. 242–43. Bibliography, pp. 330–336.

† Liefmann, H., and Lindemann, A., Der Einfluss der Hitze auf die Sterblichkeit der Saüglinge in Berlin und einigen anderen Grossstädten, Viertljhr. f. Off. Gesundhtspflg, xliii (1911), pp. 333–375.

‡ Schereschewsky, J. W., Heat and Infant Mortality, Proc. American Association for Study and Prevention of Infant Mortality, 4th Annual Meeting (1913) pp. 99–132. Reprinted in the Public Health Reports (Reprint No. 155), xxviii, No. 49 (Dec. 5, 1913).

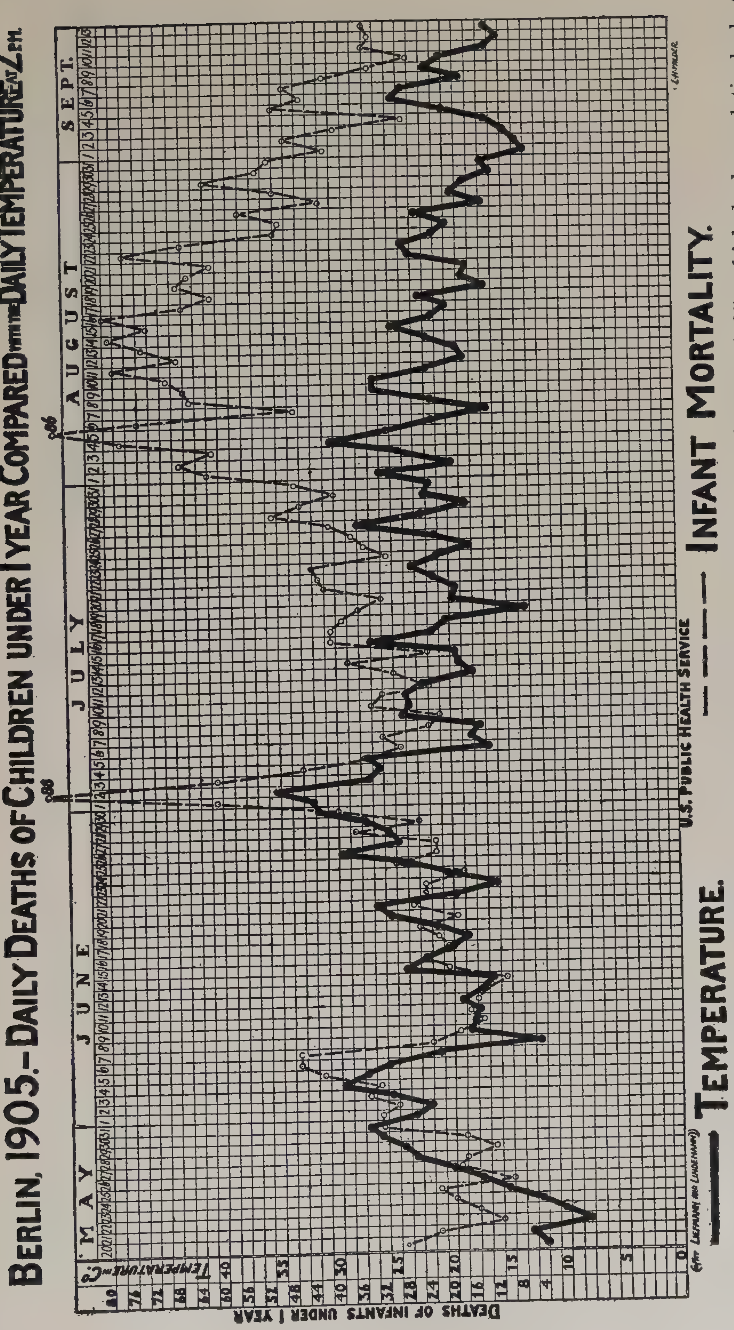

Fig. 14.—Relation of daily infant deaths to daily temperatures in Berlin, during the year 1905, which had a relatively hot summer. See: Schereschewsky, J. W., Heat and Infant Mortality, Proc. 4th Annual Meeting A. A. S. P. I. M. (1913), p. 102.

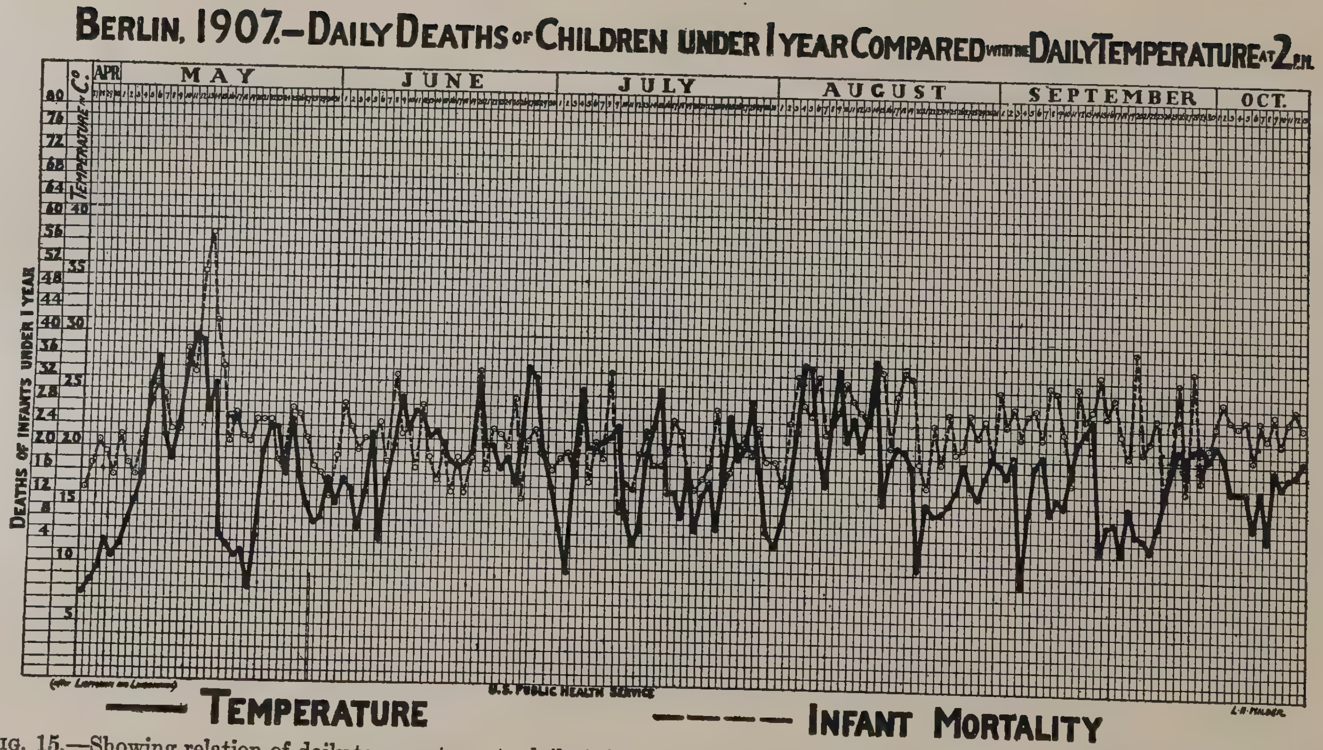

FIG. 15.—Showing relation of daily temperatures to daily infant deaths in Berlin during a cool summer, 1907. *Ibid.*, Fig. 14, p. 104.

known increase in the number of their deaths which takes place in the summer months. No incidence of death in any other age group seems to be so immediately determined by meteorological conditions." His conclusions are so cogent that it is well to state them here:

"1. The action of heat as a direct cause in the summer mortality of infants has been greatly underestimated in the last 25 years. In the future much more weight should be given to its influence.

"2. The lethal action of heat is a function, not so much of the maximum and mean temperatures of the external air as of the indoor temperatures which, in the late summer, may continue to be high, in spite of remissions in temperature of the external air.

"3. The action of dirty and stale milk in causing the death of infants has been given a significance which has overshadowed other factors of equal or greater importance.

"4. There is evidence to show that a certain proportion of infant deaths are due to specific infections, in the dissemination of which contact infection and flies doubtless play a part.

"5. As a result, future activities for the prevention of infant mortality must concentrate themselves to a greater extent on the question of housing, especially the conditions productive of high indoor temperatures, such as overcrowding, narrow streets and the absence of thorough ventilation.

"6. Poor housing conditions can be partially neutralized by the proper care of babies in the summer. The general public should be educated as to the importance of high indoor temperature in causing the death of infants, and especially as to measures which prevent babies from suffering from the heat.

"7. Breast feeding must still be regarded as a most, if not the most, important preventive of the summer death of infants."*

Heat, therefore, has both a direct and indirect bearing upon the summer mortality of infants. The heat may be so intense at the beginning of the summer that a certain number of babies will suffer from primary heat stroke and succumb before anything can be done to save them. This happens even to normal breast fed babies, especially to those of the tenement districts where the indoor temperatures are high and do not fall during the night. If the hot spell is prolonged, more and more babies loose their resistance. If artificially fed a gastro-intestinal disturbance may intervene and lead to serious results. Even after the hot wave recedes the infant deaths may remain unduly high.

Hot weather hygiene for infants is now well recognized. Through the instruction of public health nurses in the homes and the broader educational campaign of the press it has been possible to cut down considerably the summer deaths due directly or indirectly to heat.

Economic Condition.—A number of careful studies have been made abroad and in this country to determine the relation of economic and social status to infant mortality. While the methods of investigation have differed and the completeness of the data been variable, there has been general agreement that the income of a family has a distinct bearing upon the infant mortality within the family group. The infant mortality studies of the Children's Bureau in a number of American cities indicate this clearly as is seen in the following table showing the infant mortality rates according to the father's earnings (Table 16). The condition of well-being or of poverty in a family not only manifests itself in a decreasing or increasing infant mortality, but is reflected also at higher ages. This has been well shown by Concetti* for Italian families (Table 17).

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* *Ibid.*, p. 125.

TABLE 16.—INFANT MORTALITY RATES BY FATHER'S EARNINGS*

City	Deaths of infants under one year of age per 1,000 live births, by specified annual earnings of father		
	All earnings	$1250 and over	Under $550
All cities	111.2	64.3	151.4
Johnstown	130.7	87.6	260.9
Manchester	165.0	58.3	204.2
Brockton	96.7	73.5	67.1
Saginaw	84.6	22.2	142.0
New Bedford	130.3	59.9	168.7
Waterbury	122.7	68.4	151.1
Akron	85.7	40.0	117.5
Baltimore	103.5	64.7	138.0

* Compiled from Infant Mortality Studies of the Children's Bureau, U. S. A.

TABLE 17.—FROM CONCETTI†

	Per cent. of total deaths		
	Wealthy	Middle class	Working class
Deaths from 0 to 1 year	8.9	17.5	30.5
Deaths from 1 to 2 years	1.9	5.5	11.5
Deaths from 3 to 5 years	2.6	6.5	13.6
Deaths from 6 to 10 years	1.3	3.8	6.8
Deaths from 11 to 14 years	0.8	1.1	2.5

When we study the mortalities of infancy in relation to the father's occupation confirmatory evidence of the close relation of economic and social status to infant mortality is brought out. This can be tabulated from figures presented in the Report of the Registrar-General for Births, Deaths and Marriages in England and Wales for the year 1911 as follows:

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† Concetti, Luigi, L'Igiene del Bambino Seconda Edizione, Roma (1914), Tabella XIV, p. 12.

TABLE 18

OCCUPATION OF FATHER	INFANT MORTALITY, DEATHS UNDER ONE YEAR PER 1,000 LIVE BIRTHS
Upper and middle class	76.4
Agricultural laborers	96.9
Shopkeepers, dealers, etc	106.4
Skilled workmen	112.7
Intermediate workmen	121.5
Textile workers	148.1
Unskilled workmen	152.5
Miners	160.1
Total under one year (1911)	130.0

Back of these figures are to be found not only differences in social position but many other closely interwoven factors. Ignorance and poverty, as has so often been pointed out, are basic factors in the infant mortality problem. The extension of general education, especially along lines of sanitation and hygiene, and the maintenance of a sound national economic life should do much in keeping down the infant mortality rate.

Prenatal Care.—From this brief consideration of the more important contributing causes of infant mortality let us turn to some of the direct causes and see what can be done to meet them by public health measures. The seriousness of the prenatal deaths has already been mentioned. In writing upon "Certain Neglected Aspects of the Problem of Infant Mortality," Van Ingen has said that, "while a great deal has been done for babies who are old enough to be watched from prophylactic dispensaries and from milk stations, very little organized effort has been made until recently to prevent those deaths due to ante-natal influences. In 1908 the Pediatric Department of the New York Outdoor Medical Clinic undertook the systematic care of expectant mothers registered at this clinic. This work has been under the care of Dr. Herman Schwartz, and has been carried on in a most efficient way. Since 1909 the Committee on Social Service of the Women's Municipal League in Boston have carried on a campaign of prenatal instruction. Their patients have all been women who were confined by a physician or in hospitals. No cases who were to be attended by midwives were accepted. In most cases a small fee was paid by these women. A certain amount of care of expectant mothers has also been carried on since 1908 by the New York Association for Improving the Condition of the Poor, through their Caroline Rest nurses."*

In the summer of 1911 the New York Milk Committee initiated an experiment in prenatal nursing and instruction. A corps of trained public health nurses who were given very definite instructions in prenatal care were placed in a district in Manhattan. These nurses visited pregnant mothers referred to them from various sources. The women were urged to keep in close touch with their physician or a maternity dispensary. This experiment proved so successful that it was taken over by the city Bureau of Child Hygiene and eight full time nurses placed on prenatal work.

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* Van Ingen, Philip, New York Medical Jour., xiii, No. 11 (Nov., 1913), pp. 605–607.

Perhaps the most completely developed plan of prenatal service is that of the Maternity Center Association of New York City. The set-up of the service has been fully described by Miss Anne A. Stevens, its Director, and is given here in her own words:

"At the suggestion of a committee of obstetricians, Manhattan was divided into ten zones, and it was planned to establish maternity centers and substations in each of these ten zones; each center to be the focus of an educational campaign for maternity care for that zone, to conduct doctor's clinics where medical supervision will be given all patients who have not engaged their own physician or registered at a hospital until such time as they can be persuaded to do so; the nurse in charge of each center together with nurses in coöperating clinics to reach practically every pregnant mother in the zone, to teach her the need for medical and nursing care throughout pregnancy, teach her what and how to prepare for her baby, help her arrange for her care at time of confinement and keep in close touch with her until she really knows how to care for her baby. It was planned that each center consist of an examining room where a doctor's clinic could be held once a week or oftener, a dressing room for patients in order to assure them privacy and a waiting room, made as nearly like a comfortable sitting room as possible, where there could be a continuous exhibit of a model baby's bed, layette, toilet tray, etc., and a bed properly made for the mother's delivery. This work was to be financed and directed by this voluntary organization of citizens called the Maternity Center Association, only until such time as a demonstration could be made so convincing as to assure an adequate appropriation of public moneys to carry it on.

"When the association was formed, the New York Milk Committee took the entire responsibility for the work in two of the zones. The Women's City Club continued to finance the work in the zone where they established the first Center, but put the actual nursing under the direction of the Maternity Center Association."*

The results of the prenatal care given by the Maternity Center Association of New York and the Instructive District Nurses Association in Boston are so striking that they were presented in graphic form in the Report of the Special Commission to investigate Maternity Benefits in Massachusetts, and are here reproduced (Figs. 11 and 12).

Institutional Mortality.—The infant mortality in institutions erected for their care has always been excessively high. In the seventeenth and eighteenth century foundling asylums of Europe the mortality ranged from 75 to 100 per cent. among babies in their first year. Some few institutions by extreme care and individual attention with the aid of wet-nurses were able to reduce the mortality somewhat, but even at the best many more babies died in institutions than died among those who were left with their own mothers or placed out in boarding homes. During the twentieth century the institutions have been greatly improved so far as sanitary conditions are concerned. More individual attention is given, and the feeding is scientifically carried out. In a few of the modern institutions for foundlings and abandoned babies a surprisingly low mortality rate is maintained, especially among those who have passed the first few perilous months. The neonatal mortality in institutions is still quite high, but in properly conducted lying-in hospitals, where suitable prenatal care has been given, the early mortality has been greatly reduced. One of the best studies of institutional mortality of the new-born is that by Holt and Babbitt based upon a careful analysis of 10,000 consecutive births at the Sloane Maternity Hospital in New York.* They found that the neonatal deaths during the first 14 days in the hospital numbered 3 per cent. of the living births, and that for 50 per cent. of this number "prematurity" could be assigned as a cause of death. Forty-eight per cent. of the total deaths and 66 per cent. of the premature deaths occurred on the first day. "Congenital weakness" and "atelectasis" were given as a cause of death in 58 per cent. of the total deaths. Accidents of birth made up about 20 per cent. of the deaths in the first 14 days. Syphilis was given as a cause of death in 4 per cent. of the cases, and played a large part in the still births.

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* Stevens, Anne A., The Work of the Maternity Center Association, Trans., 10th Annual Meeting of American Child Hygiene Assoc. (1919), pp. 43–63.

The consensus of opinion of those who have studied the institutional problem in relation to child welfare is that the fewer days spent in an institution the better. Every effort should be made to keep the baby with its own mother, but where this is absolutely impossible well selected and carefully supervised boarding homes for babies ought to be provided. The ideal "home" should not take more than two babies and the compensation given should make it possible for the babies to secure the best of care. Not only should such "homes" be thoroughly supervised by the health authorities, but the babies in them be taken regularly to an infant welfare center for periodic examination and adjustment of their feeding.

Syphilis.—A searching study of the early deaths reveals that congenital syphilis is a cause in more than the death returns seem to indicate. Many deaths from syphilis are undoubtedly obscured in our vital statistics by being simply reported as "still births," "premature births," "congenital debility," etc. Every still birth should be carefully registered and if at all possible its exact cause determined. Such an examination of the still births occurring in maternity hospitals and out-patient work has shown that syphilis is probably the largest cause of still births. The recent work of Williams at Johns Hopkins and Jeans at Washington University in St. Louis confirm our earlier conclusions and point to syphilis as not only an important cause of still births, but of a considerable number of deaths of full term infants as well. By Wassermann reactions applied to the maternal, placental and fetal bloods and a careful study of the placentæ for syphilitic lesions and spirochetæ pallida it is now possible to gain a much fuller picture of syphilis of the newborn.

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* Holt, L. Emmett, and Babbitt, Ellen C., Institutional Mortality of the Newborn, Proc. A. A. for S. and P. of Infant Mortality, 5th Annual Meeting (1914), pp. 151–160.

In a critical study of 302 fetal deaths occurring in 4,000 consecutive deliveries at Johns Hopkins Maternity between April, 1916, and December, 1919, Williams* found the following (212 of the dead babies coming to autopsy):

TABLE 19

Cause of death	Number of cases	Percentage
Syphilis	104	34.44
Dystocia	46	15.20
Toxemia	35	11.55
Prematurity	32	10.59
Cause unknown	26	8.61
Placenta previa and premature separation	16	5.28
Deformity	11	3.64
Eleven other causes	32	10.69
	302	100.00

Of the 4,000 women studied as regards syphilis 421 gave a positive Wassermann reaction. These patients were grouped in three classes: (*a*) No treatment, (*b*) inefficient treatment, the patients who received two or three salvarsan injections but no after treatment, and (*c*) satisfactory treatment, "the patients received from four to six injections of salvarsan followed by a course of mercurial treatment, with the result that the Wassermann became negative and remained so." The results are shown in the following table:

TABLE 20

TREATMENT	NUMBER OF PATIENTS	PER CENT. OF CHILDREN BORN DEAD OR PRESENTED SOME EVIDENCE OF SYPHILIS
(*a*) No treatment	157	52.0
(*b*) Inefficient	103	37.0
(*c*) Satisfactory	163	7.4

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* Williams, J. Whitridge, The Significance of Syphilis in Prenatal Care and in the Causation of Fetal Death, Bulletin of the Johns Hopkins Hosp., xxxi, No. 351 (May, 1920), p. 143.

Williams therefore concludes that "the evidence at our disposal shows that if syphilis is recognized early in the pregnant woman, and is intensively and appropriately treated, almost ideal results may be obtained so far as the child is concerned. Consequently, there is every reason to hope that in the future syphilis may be practically eradicated as the cause of fetal death in all properly conducted clinics in which the women register prior to the middle of pregnancy."*

An exhaustive review of the literature of syphilis as it bears upon infancy and childhood has been presented by Jeans.† He has also made a careful study of syphilis in its relation to infant mortality. Those interested in this important phase of the subject should consult the work of Jeans. He very properly points out that we must take into consideration not only the infant mortality from this devastating disease but also its prevalence among all individuals of the procreating age. In other words, syphilis is eminently a "social disease" and its social roots must be discovered and grubbed up before we can hope to eliminate its influence upon the mortalities of infancy and childhood. In order to show how widespread syphilis is among our general population and to bring out how this affects the infant mortality the following tables from Jeans are given:

TABLE 21.—WASSERMANN SURVEY AMONG ADULTS ADMITTED TO HOSPITALS AND DISPENSARIES‡

City	Number examined	Number having positive Wassermann	Per cent. having positive Wassermann
Baltimore	1,080	116	10.8
Boston	4,000	600	15.0
Chicago	418	56	13.4
Ann Arbor	2,771	160	5.8
San Francisco	6,995	518	7.4
Total	15,264	1,450	9.5

In an examination of 11,933 accepted U. S. Army recruits Vedder found 13 per cent. gave a positive Wassermann and among 856 candidates for the police force in Washington, D. C., 15 per cent. were positive.

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* *Ibid.*, pp. 144–45.

† Jeans, Philip C., American Jour. of Diseases of Children, xx (1920), pp. 54–74 and 132–152.

‡ Jeans, Philip C., Syphilis and Its Relation to Infant Mortality, Trans. Am. Assoc. for Study and Prevent. Inf. Mortality, 9th Annual Meeting (1918), pp. 146–156, Tables I and II.

TABLE 22.*—WASSERMANN SURVEY AMONG MARRIED PREGNANT WOMEN

CITY	NUMBER EXAMINED	PER CENT. HAVING POSITIVE WASSERMAN
Chicago	116	10.6
Brooklyn (1)	1822	8.0
Brooklyn (2)	892	7.9
Philadelphia	40	7.5
New York	2488	11.5
Total	5358	9.66

* *Ibid.*, Table II.

The bearing of this high percentage of syphilis in adults of child-bearing age upon fetal deaths is brought out in the following table:

TABLE 23.†—SHOWING THE FREQUENCY OF FETAL DEATH IN SYPHILITIC FAMILIES

Authorities	Families	Total pregnancies	Miscarriages and still births	Per cent.
Holt	193	427	123	28.8
Love	21	172	32	18.6
Harman	150	1001	172	17.2
Jeans	100	331	131	40.0
Jamieson	71	253	95	37.5
Hochsinger	134	569	253	44.4
Raven	82	350	101	28.9
Post	30	168	53	31.6
Haskell	58	167	44	26.2
Tarnier	42	90	56	62.0
Julien	...	206	44	21.4
Pileur	...	414	154	37.0
Totals	...	4148	1258	30.3

† *Ibid.*, Table III.

From this mass of statistics, and others referred to in the original communications, Jeans draws the conclusions:

"1. From 10 to 20 per cent. of adult males and about 10 per cent. of married women are syphilitic and a minimum of 10 per cent. of marriages involve a syphilitic individual.

"2. Seventy-five per cent. of all the offspring in a syphilitic family are infected.

"3. In a syphilitic family 30 per cent. of the pregnancies terminate in death at or before term, a waste three times greater than is found in non-syphilitic families.

"4. Thirty per cent. of all the living births in a syphilitic family die in infancy as compared to a normal rate of 15 per cent. in the same class.

"5. Probably 25 to 30 per cent. of clinically syphilitic infants die as a result of syphilis.

"6. But 17 per cent. of all the pregnancies in syphilitic families result in living non-syphilitic children who survive the period of infancy.

"7. About 5 per cent. of our infant population is syphilitic.

"8. According to St. Louis vital statistics, 3.5 per cent. of all infant deaths are ascribed to lues."*

Jeans entertains essentially the same opinion as Williams that considerable can be done in the way of treatment of the pregnant mother in preventing the large number of fetal and neonatal deaths which we now see from this cause. In order to be effective the treatment must be begun early and continued over a sufficient period. Only through a well organized prenatal service can this be carried out. In all dispensary and hospital practice the Wassermann reaction should be a routine procedure. After treatment is started a thorough follow-up of each case is necessary. Here, as is the case with tuberculosis, the application of social and economic measures is all important.

Tuberculosis.—Our knowledge of tuberculosis in childhood has been considerably extended during recent years. The routine application of the von Pirquet test, the use of the Roentgen rays and more searching physical diagnosis have gradually shifted the emphasis from the tuberculous adult to the tubercularized child. It is now generally held by those who have carefully studied the results of our modern research that tuberculosis is largely a childhood infection by direct contact with open cases of tuberculosis. There is no question that tuberculous milk may infect the child but in those countries where special attention is given to the milk supply, and pasteurization or boiling is almost universal, the danger from milk infection is very slight. In England, however, milk infection appears to be quite frequent and in Edinburgh, according to Thomson and other observers there, the principal means of childhood infection.

The incidence of childhood infection in various localities differs considerably, but there is general agreement that congenital tuberculosis is comparatively rare; that infection takes place largely after birth in the home environment during the first year and in the wider social environment after infancy; that there is an increasing proportion of infection with the increase in age of the children until at puberty from 70 to 90 per cent. have received a tuberculous infection. We are all familiar with the classical studies of von Pirquet, Hamburger and A. Ghon. Some of von Pirquet's† early figures are here given to compare with our present ones, as they confirm the soundness of his original investigations. von Pirquet obtained the following results from tests upon 1407 children seen at the Escherich Clinic, Vienna:

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* *Ibid.*, p. 153.

† von Pirquet, Clemens, Frequency of Tuberculosis in Childhood, Jour. A. M. A., (Feb. 27, 1909), lii, pp. 675–678.

TABLE 24

Years	0–1	1–2	2–3	3–4	5–6
Number of all cases	410	116	98	110	91
Reaction positive	21 (5%)	24 (4%)	32 (33%)	42 (38%)	50(55%)
Manifestly tuberculous reacting	21	23	19	23	18
Manifestly tuberculous not reacting (miliary tuberculosis)	1 (5%)	4 (20%)	1 (19%)	3 (21%)	1 (20%)

Positive reaction in 80 per cent. of the cases was reached in the tenth year. von Pirquet remarks that the percentage of reacting children is particularly high in his tables because "tuberculosis is notoriously prevalent in Vienna," especially among the poorer classes. When we compare the results of others with those given above we find that von Pirquet's figures are not so extraordinary after all.

The following table from Fishberg showing the ages of the children and the number and percentage of children giving positive and negative von Pirquet reactions is instructive:

TABLE 25.—PERCENTAGE OF CHILDREN REACTING TO TUBERCULIN TEST (VON PIRQUET) ADMINISTERED ONCE (NEW YORK). (AFTER FISHBERG)

Age	Number	Positive		Negative	
		Number	Per cent.	Number	Per cent.
Under 6 months	22	1	4.5	21	95.46
6 to 12 months	34	5	14.71	29	85.29
2 years	39	13	33.33	26	66.67
3 years	36	14	38.89	22	61.11
4 years	44	19	43.18	25	56.82
5 years	51	24	47.06	27	52.94
6 years	55	29	52.73	26	47.27
7 years	45	27	60.00	18	40.00
8 years	45	28	62.22	17	37.78
9 years	40	27	67.50	13	32.50
10 years	43	30	69.77	13	30.23
11 years	35	22	62.86	13	37.14
12 years	44	29	65.91	15	34.09
13 years	35	27	77.14	8	22.86
14 years	20	15	75.00	5	25.00
Totals	588	310	52.72	278	47.28

Although the incidence of tuberculous infection in the first year of life is not very high it is sufficiently serious to merit our attention in the prevention of infant mortality. The babies infected early exhibit but little resistance to the disease and most of them perish within the first year from a generalized tuberculosis or tuberculous meningitis. In the year 1919 the death returns in the Registration Area of U. S. gave 0.7 per cent. of the total deaths under one year as due to tuberculous meningitis, 0.5 per cent. to tuberculosis of the lungs and 0.2 per cent. as due to other forms of tuberculous. In other words 1.5 per cent. of the total deaths in infancy in the Registration Area were due to tuberculosis, and this may be taken as a conservative estimate as a number of babies reported dying from "convulsions," "malnutrition," "bronchitis," etc., undoubtedly suffered from a "concealed tuberculosis."

We may, therefore, sum up our present knowledge of tuberculosis as it relates to childhood as follows:

1. Tuberculosis is not hereditary in the strict sense of the word.
2. Congenital tuberculosis is rare.
3. Tuberculous infection takes place in the child, the incidence of infection being small the first year and gradually increasing with each year of childhood.
4. Tuberculosis in infancy develops rapidly into an active form, and the mortality during the first year of life is very high.
5. Direct contact with tuberculous adults is the chief mode of infection in infancy, although tuberculous milk may play a part in communities where breast feeding is not prevalent and the general milk supply is not pasteurized or boiled.
6. In the future anti-tuberculosis measures must be directed more to the prevention of infection in childhood, and the building up of the child's resistance.
7. Exposure to minimal doses of tubercle bacilli produces a relative immunity.
8. We still look for a specific immune sera or vaccine which will assist the body in acquiring and preserving its immunity.

The Acute Communicable Diseases.—Although the acute communicable diseases of childhood make up only about 4 per cent. of the total number of deaths under one year they are of sufficient importance to demand careful consideration. Whooping-cough and measles continue to come in recurring epidemic waves and take a heavy toll of infant lives. Very little progress on the whole has been made in the prevention of these two diseases. The complications and sequelæ of both leave a damaged childhood which we try to patch up in later years. To give some idea of the seriousness of the four important contagious diseases of childhood and the period of childhood in which they take their largest toll the diagram showing the experience in Ohio for 10 years is given (Fig. 16).

One of the most instructive studies in this country on the morbidity and mortality of pertussis and measles is that of Veeder.* A painstaking statistical study of measles was also carried out by the late Frederick S. Crum.† Vedder points out that while the death rates for diarrhea and enteritis, tuberculosis, diphtheria and typhoid have decreased in the last 15 years, those for measles and whooping-cough remain practically the same. The number of deaths from each of these diseases each year in the United States is in the neighborhood of 10,000. Of this number about 80 per cent. of the whooping-cough and 50 per cent. of the measles occur in children under two years of age. Crum* estimates that a little over 1 per cent. of all deaths in the temperate zone are due to measles. The charts from Veeder show the distribution by age of the deaths from the four principal communicable diseases of childhood (Figs. 18 and 19).

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* Veeder, Borden S., The Morbidity and Mortality of Pertussis and Measles with Particular Reference to Age, Proc. American Assoc. for Study and Prevent. Infant Mortality, 7th Annual Meeting (1916), pp. 86–101. Charts and diagrams.

† Crum, Frederick S., A Statistical Study of Measles, Amer. Jour. Pub. Health, iv, No. 4 (1914), pp. 289–309.

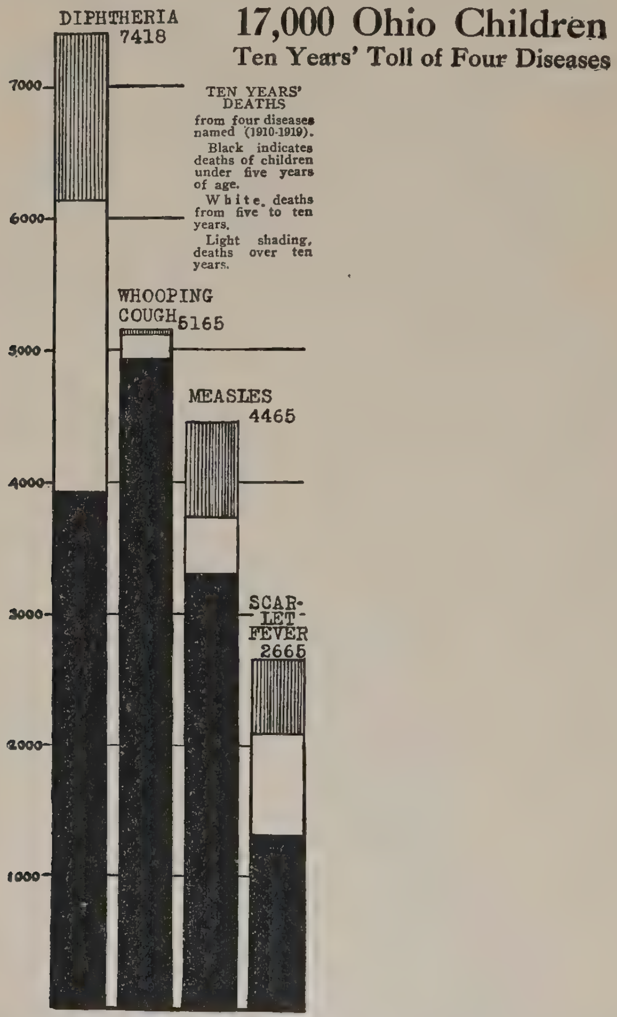

FIG. 16.—Serious loss of life under five years of age from four important diseases. (From The Ohio Public Health Journal, xi, No. 12, Dec., 1920, p. 195.)

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* *Ibid.*, p. 290.

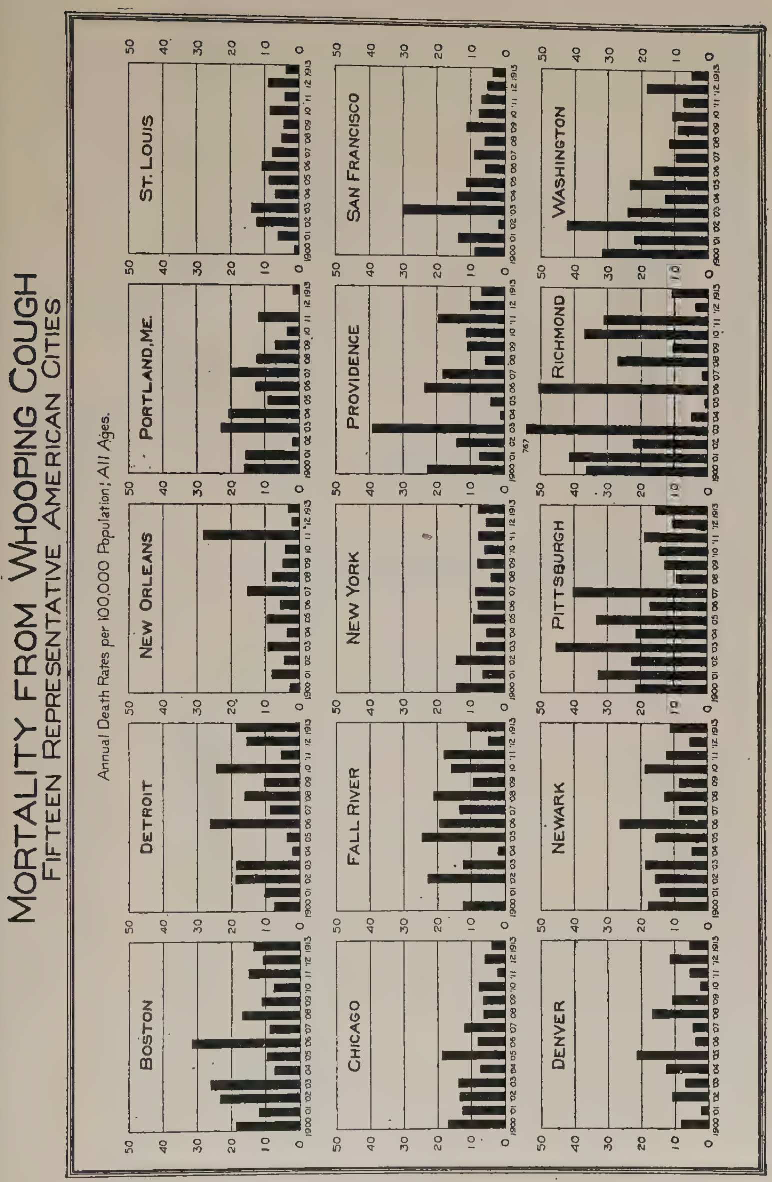

FIG. 17.—From The American Journal of Public Health, 19, p. 1011.

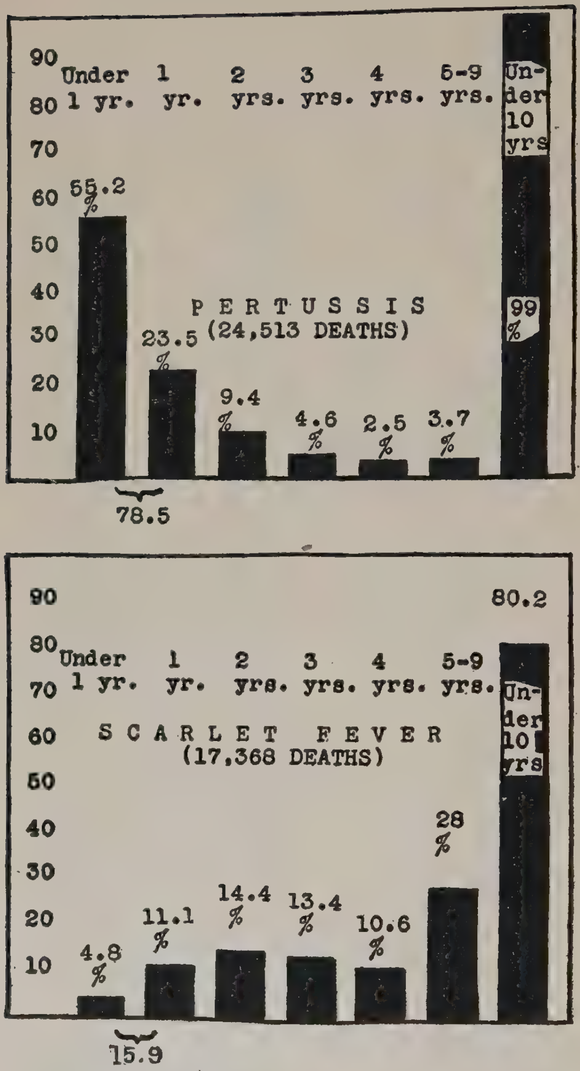

FIG. 18.—Distribution of Pertussis and Scarlet Fever deaths by age. U. S. Registration Area, 1910–13. (From Veeder.)

Whooping-cough is even more fatal for children under one year of age than measles, or any other of the acute communicable diseases. The fatal termination of this disease is usually, as is the case with measles, due to one of its complications, most often of the respiratory tract. The mortality from whooping-cough varies in different cities (Fig. 17), as is seen from the chart presented by Crum. The mortality from whooping-cough in the Registration Area of the United States according to age and sex and age distribution of deaths is well brought out in these charts (Fig. 20).

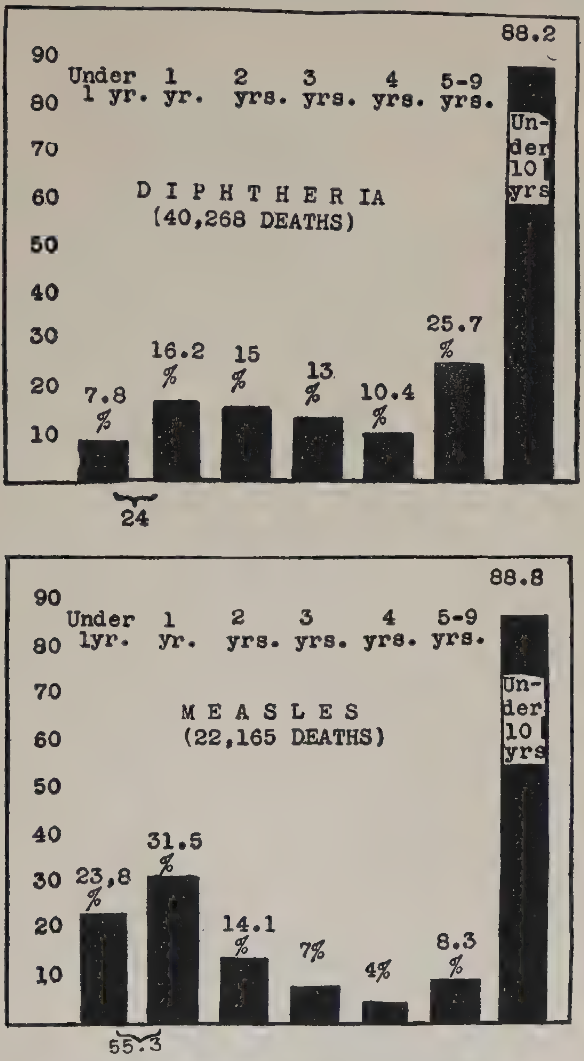

FIG. 19.—Distribution of Diphtheria and Measles deaths by age. U. S. Registration Area, 1910–13. (From Veeder.)

The prevention of both measles and whooping-cough is exceedingly difficult and the same reasons to a large extent apply to each. Abt,*

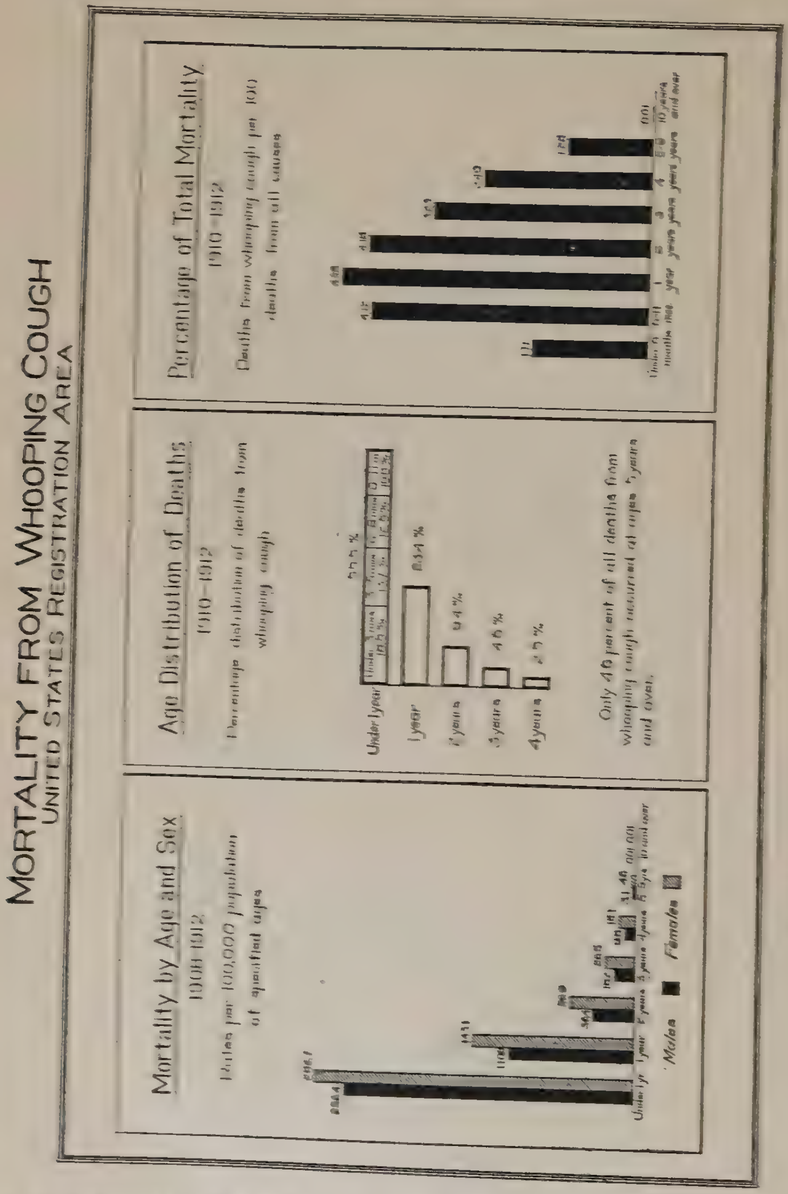

FIG. 20.—From American Journal of Public Health, 19, p. 1007.

in an admirable study of pertussis, has pointed out the difficulties of its prevention under the following heads:

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* Abt, Isaac A., Pertussis, Proc. Amer. Assoc. for Study and Prevent. of Infant Mortality, 7th Annual Meeting (1916), p. 134.

"1. The disease is infectious during the early stages.

"2. The disease presents so few symptoms at the onset that the diagnosis can be positively made only after a lapse of considerable time when definite symptoms occur.

"3. On account of the mildness or atypical symptoms the true nature of the disease sometimes remains unrecognized during the entire course.

"4. In spite of an evident diagnosis the children are sometimes sent to school.

"5. Convalescents are sometimes admitted to their classes before the contagiousness of the disease has disappeared."

Our next great advance in the prevention of the contagious diseases of childhood must be the prevention of infant infection from whooping-cough and measles and in the immunization against diphtheria. The respiratory diseases of infancy are so closely related to whooping-cough, measles, and tuberculosis that means taken to prevent these infections will have a marked effect upon deaths from bronchitis and pneumonia. More attention should be given to the "common cold" as a caustive factor in the acute respiratory diseases of infancy. Before much headway in the prevention of these contagious diseases can be brought about a wider educational publicity must be conducted to reach the mothers and fathers in the homes. Without a keen civic conscience regarding the spread of the communicable diseases little can be expected through isolation and quarantine feebly enforced by the health authorities.

Historical Resumé of Child Hygiene.—The historical development of the study and prevention of infant mortality and of child welfare work has been so thoroughly and delightfully treated by Holt in his Address on "Infant Mortality, Ancient and Modern: An Historical Sketch," from which many of the facts used in this summary are drawn, and by Garrison as a part of his chapter on the "History of Pediatrics" in Volume I that it will be necessary here simply to touch briefly some of the outstanding peaks of progress in maternity and child hygiene as it has extended from country to country. For convenience of reference these will be noted chronologically with but a short statement as to their historical and practical importance:

1817. John Bunnell Davis' classical little book entitled, "A Cursory Inquiry into Some of the Principal Causes of Mortality among Children, with a View to Assist in Ameliorating the State of the Rising Generation in Health, Morals and Happiness." London, 1817. This book was strangely modern in stating many of the underlying principles which we stress today. The importance and duty of breast feeding were strongly urged.

1844. First *crèche*, or day-nursery, established in Paris by M. Firmin Marbeau, Mayor of the first arrondissement. This was designed for poor working mothers to leave their infants while they went out to work. It was presided over by a nun, and a visiting physician called daily. From 1844 to 1847 nine more crèches were established in Paris.

1847. Société des Crèches formed in Paris to bring about better coöperation and standardize methods. By this time the idea of the crèche was taking hold on other parts of Europe and the number of crèches established in other countries increased rapidly.

1861. Infant Mortality recognized as a social problem at the meeting of the National Society for the Promotion of Social Science held at Dublin. Sir William Moore delivered an address on "Some of the More Prominent Causes of Excessive Infant Mortality" in which he pointed out clearly the socio-economic factors.

1865. Société Protectrice de l'Enfance organized in France to encourage breast feeding; supervise babies placed out to wet-nurse and instruct mothers in child hygiene. After 10 years of successful work the Society was able to show good results in the reduction of infant mortality in the district it touched.

1865. Dr. Wm. Farr considered "Infant Mortality" in his Annual Report of the Registrar-General of England and Wales.

1871. Infant Mortality discussed at a meeting of the Social Science Association in Philadelphia. An Address by John S. Parry on "Infant Mortality and Necessity of a Foundling Hospital in Philadelphia."

1872. Life Protection Act in England designed to protect "farmed-out" babies by requiring registration and licensing of all boarding homes for infants.

1873. New York Diet Kitchen Association formed and a milk station established.

1874. *Loi Protectrice des Enfants*, better known as the Roussel Law, was passed in France to ensure supervision of children under two years of age placed out to nurse, and of the boarding homes where they were placed. Registration, licensing and inspection of all places, where babies were boarded out away from their parents, were provided for in this law.

1876. *Société d'Allaitement Maternelle*, a Society for nursing mothers, was organized in Paris primarily for the encouragement of breast feeding. It aimed to encourage maternal nursing by providing for both the expectant and nursing mother. It sent friendly visitors into the homes to assist mothers during the prenatal period and after confinement, and offered facilities for the mothers to consult a physician from time to time during pregnancy. Prematernity homes were established. Maternity grants were allowed. Regular monthly observations of the infants by physicians and home visitors were provided. During 16 years the Society cared for about 10,000 mothers in prematernity homes with excellent results. The prenatal service showed marked results in the reduction of maternal and neonatal mortality. It is reported that there were no deaths of the mothers who were received into the maternity hospitals from the prematernity homes. Pinard claimed that the babies were in better condition and weighed more than others not thus cared for.

1876. Infant Mortality considered at the First International Congress of Hygiene in Brussels. Mortalité des Enfants Nouveau-Nés, Congrès International d'Hygiène, Vol. 1, Brussels, 1876.

1877. First law prohibiting pregnant women from working in factories a certain number of weeks before and after confinement was passed in Switzerland. Similar laws were passed in Hungary (1884), Austria (1885), Holland (1889), Belgium (1889), England (1891), Germany (1891).

1878. Second Meeting of the International Congress of Hygiene in Paris. At this meeting a committee consisting of Bertillon, Marjolin and Bergeron gave a comprehensive report on the causes, extent and prevention of infant mortality.

1890. First simple consultation for nurslings opened by Professor Herrgott at Nancy, France. This was called "L'Oeuvre de la Maternite."* Infants born at the Maternity Hospital were required to be brought back one month after birth for a complete medical examination, and if found in good condition and gaining favorably the mothers were given a money subsidy. During the years 1890–1900, 2,052 women were cared for in that institution and 25,382 francs were distributed. The results proved uniformly good.

1892. First fully developed "Consultation de Nourisson" opened in June by Dr. Pierre Budin in connection with the Charité at Paris. Its prime object was the supervision and hygienic direction of infants born at the Maternity Hospital. Great stress was laid upon the establishment and continuance of breast feeding. Regular medical supervision was kept up for one or two years with frequent visits of the mothers with their babies to the "Consultation."

1892. In July Dr. Henri Variot established in Paris a milk station in connection with the Belleville Dispensary, called "Distributation de Lait au Dispensaire de Belleville." This has also been called by some "L'Oeuvre de la Goutte de Lait" and said to be the first "Goutte de Lait," but generally the first "Goutte de Lait" is attributed to Dr. Dufour of Féchamp.

1893. Nathan Straus Infant Milk Depots established in New York City where pasteurized milk modified according to certain fixed formulæ was dispensed from six milk depots at a reduced cost.† Over 2,300 babies were fed on the Straus modified and pasteurized milk daily during the first year and between 4,000 and 5,000 babies were benefited during that year.†

1894. The first "Goutte de Lait" as a separate institution, outside the metropolis, was established by Dr. Leon Dufour in the small Norman town of Féchamp, and he was the first to give the name "Goutte de Lait" to these "milk stations." As stated by Dufour the objects of the "Goutte de Lait" were:

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*See: Herrgott, Annales de la Société Obstétricale de France, 1901.

†Straus, Lina Gutherz, "Disease in Milk—The Remedy Pasteurization." The Life and Work of Nathan Straus (1917), pp. 75 ff., E. P. Dutton and Co., New York.

"1. To induce mothers by advice and by every possible encouragement to feed their babies at the breast.

"2. When it is impossible for the child to be wholly breast-fed, the mother is urged not to abandon breast feeding altogether, but to supplement her own milk with suitably prepared cow's milk, so as to secure that the infant shall be, if not wholly, at least partially breast-fed.

"3. When, however, there is no doubt that it is impossible, physically, socially, or morally, for the mother to suckle her baby, the Goutte de Lait endeavors to secure that the infant shall not lack maternal care and affection. For this purpose there is given under the best possible conditions a milk of good quality, with advice as to the conditions necessary for success in artificial feeding."*

These local "milk depots" for combating infant mortality were models for others established throughout continental Europe and England and transplanted, with certain modifications, to America where they later developed into our infant welfare centers.

1894–1903. Villiers de Duc Demonstration in France. Mayor Morel, basing the demonstration upon the experience of his father in the same village 1854–1863, devised a simple but effective scheme for the saving of infant life and enforced it through his office as Mayor. Every pregnancy was reported early. Breast-feeding was encouraged for every baby. Every case of illness in a mother or child was reported within 24 hours and medical help was made available. Regular consultations were held, and a good milk supply assured those who could not nurse their babies. This program was carried out for 10 years with signal success, as it is reported that not a single mother or baby died in the village during that time.†

1895. Consultation de Nourrissons established by Budin at Maternité Hospital in Paris.

1896. National Congress for Infancy at Florence, Italy.

1897. Milk Station established at Rochester, New York.

1897. First Belgian "Goutte de Lait" established in Brussels by Dr. Eugene Lust. Called a "Laiterie Maternelle."

1899. Committee of the St. Helen's Town Council, England, sent to Féchamp to study the "Goutte de Lait" of Dr. Dufour. Following their report the first "Goutte de Lait" was opened in England at St. Helens. In its English environment it was known as a "municipal milk depot," and differed somewhat from its French model, although carrying out the original intent of supervision of infant hygiene and feeding. This was followed by the establishment of Milk Depots in

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*Quoted by McCleary, G. F., "Infant Mortality and Infant Milk Depots" (1905), pp. 61–62. Ref. to brochure by Dr. Leon Dufour, "Comment on crée une Goutte de Lait."

†Knox, J. H. Mason, Jr., "The Next Step in a Health Program," Mother and Child, ii, No. 9 (Sept., 1921), p. 409. See also Broadbent, Benjamin, "An Open Letter to the Mothers of Huddersfield," Daily Chronicle Printing Works, Lord Street, Huddersfield, 1911.

Liverpool (1901), Battersea (1902), Leith and Bradford (1903) and Burnley, Glasgow and Dundee (1904). The Milk Depot in England gradually broadened its functions to assume more and more medical supervision of infant hygiene with the addition of health visitors to follow-up with advice and instruction in the homes.

1899. Milk Station established at the Babies' Hospital in Newark, New Jersey, by Dr. Henry L. Coit, the "father of the Certified Milk Idea."

1901. Brooklyn Children's Aid Society established eleven Milk Stations.

1902. Midwives' Act passed in England and a Central Midwives' Board was created whose duty it was to make rules and regulations relating to midwives.

1903. Milk Stations of the Babies' Milk Fund Association of Baltimore were opened.

1905. New York Association for Improving the Condition of the Poor carried on a vigorous campaign for better milk which resulted the following year in the organization of the New York Milk Committee.

1905. *Cantines maternelles* (mothers' canteens) established in Paris for expectant and nursing mothers.

1906. The New York Milk Committee created. This was a powerful factor in stimulating better milk supply, infant welfare work, better statistics on Infant Mortality, etc.

1906. Babies' Dispensary and Hospital of Cleveland, Ohio, began its work with a clearly defined plan and a scheme for city wide infant welfare work. Its underlying principles were similar to those of the Saugslingsfüersorgestellen of Berlin. Practically all the municipal infant welfare of Cleveland had as its mainspring the work of the Babies' Dispensary and Hospital. As soon as a piece of infant welfare work had been thoroughly demonstrated and worked out it was linked up to the City Bureau of Child Hygiene or other municipal activities. It has also been instrumental in carrying out a consistent plan for the education of medical students and nurses in child hygiene work.

1906. St. Louis, Missouri, Milk Commission opened in July a milk station which bore the name of "*Goutte de Lait*," the first with this name in the United States, and patterned after the French institutions of the same name.*

1906. In Huddersfield, England, with Benjamin Broadbent as Mayor the notification of births within 36 hours was made compulsory. This was an important step in the child welfare program as it made possible the early care of mothers and infants under the supervision of the health authorities.

The application of the notification of births to infant welfare proved so beneficial that Mr. Broadbent gave his influence to the passage of the Notification of Births Act which passed Parliament the following year. By means of well planned child hygiene based upon the methods used at Villers de Duc Mayor Broadbent succeeded in reducing the infant mortality of Huddersfield from 139 to 44 per thousand births.*

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* Bleyer, Adrien, "An Infant Welfare Station in St. Louis in 1906," Jour. of the Missouri State Medical Association, xviii, No. 2 (Feb., 1921, pp. 51–52).

1906. First Extensive Exhibition for Infant Welfare in Germany held at Berlin in March, in which the Government and local health authorities took great interest.

1907. England passed the Notification of Births Act.

1907. New York Association for Improving the Condition of the Poor began prenatal work.

1908. New York City Bureau of Child Hygiene, the first in the United States, was organized under Dr. S. Josephine Baker as Director. The Bureau has continued to the present under the same Director and has been a pioneer in many lines of child hygiene in this country.

1909. In June the "Kaiserin Auguste Victoria-Haus" was opened at Charlottenburg for the prevention of infant mortality in Germany. This institution was designed to combine scientific research and practical application of measures for the welfare of infants. Its objects were stated to be:

"1. To investigate scientifically and practically by means of suitable arrangements the questions concerning the nourishing and bringing up of infants and the care of mothers.

"2. To collect material concerning infant mortality and regarding the existing arrangements and organizations for the care of infants and mothers in Germany and foreign civilized countries.

"3. To make generally known in a suitable manner by publications the results of such scientific and practical investigations as well as to publish the material collected in the field of infant and mother welfare work. Further, to give information and advice to official bodies, public and private associations and private individuals."†

1909. "Deutsche Vereinigung für Saügslingsschutz" formed in Berlin to organize the scientific and practical work for the welfare of infants being done in Germany.

1909. Organization of the American Association for the Study and Prevention of Infant Mortality (now the American Child Hygiene Association) at New Haven, Conn. The Annual Transactions of this Association are invaluable handbooks on the progress of child hygiene. Its monthly magazine, "Mother and Child," gives current news items and articles on modern developments of maternity and child welfare. The Association is actively engaged in maternal, infant and child hygiene work. About 375 local organizations are affiliated with the Association and its total membership is about 2,400.

1911. New York Milk Committee began an experiment in prenatal care by employing nurses to educate expectant mothers. This work was turned over to the Bureau of Child Hygiene in 1914 and eight nurses giving all-year round prenatal service were appointed.

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* Broadbent, Benjamin, "An Open Letter to the Mothers of Huddersfield." Also "Benjamin Broadbent, the Babies' Mayor," Mother and Child, ii, No. 7 (July, 1921) pp. 298–306.

† Report, III Internationaler Kongress für Säugslingsschutz, 1911 (Gouttes de Lait), Saugslingfürsorge in gross-Berlin, verlag von Georg Stilke, Berlin (1911), pp. 351 ff.

1912. Children's Bureau of the United States Department of Labor created and Miss Julia Lathrop appointed its Chief. This Bureau, under the same Chief, has rendered excellent service to the cause of maternity and child welfare. Its studies on Infant Mortality in typical American communities should be consulted by all interested in this subject. A wealth of information is incorporated in its reports and studies issued from time to time. It centered attention upon the child during the War in the Children's Year Campaign which stimulated local efforts for child welfare all over the country, resulting in the formation of many State and municipal Bureaus of Child Hygiene. The post-war conferences on child welfare, out of which grew the Minimum Standards for Child Welfare, were arranged and carried out by the Children's Bureau.

1914. Complete scheme for Maternity and Child Welfare for England and Wales outlined by Sir Arthur Newsholme, then Chief Medical Officer of the Local Government Board. "To encourage the adoption of schemes by local authorities, the Local Government Board of England and Wales was at that time prepared to pay grants in aid of approved local expenditures, whether by local authorities or voluntary organizations, for the salaries of health visitors or other officers engaged in child-welfare work, and for clinics, dispensaries or other institutions providing medical and surgical advice and treatment to mothers, and children up to the age of five years."* The Parliamentary grants were greatly extended during and since the War and have been the main incentive to the rapid development of child-welfare work in England and Wales and also in Scotland and Ireland.

1915. The Notification of Births (Extension) Act passed in England.

1920. Formation of the National Child Health Council in the United States. A council to assist in the coördination of child health activities.

1921. July. The Sheppard-Towner Bill for the "Public Protection of Maternity and Infancy" passed the U. S. Senate and in November passed the House and was signed by the President.

1921. Beginning of the National Child Health Demonstration at Mansfield, Ohio.

Attention Now Focused on Child Welfare.—It is no longer necessary to urge among our intelligent communities the vital need for well planned child welfare work. The World War forced upon us a consideration for our mothers and children which the slower forces of social evolution may have taken decades to do. The telling campaign of education carried on during the Children's Year by the Children's Bureau stimulated child welfare endeavors in many directions. The post-war conferences planned by the same Bureau resulted in setting up certain minimum standards of Child Welfare. The experience of the war torn countries, notably of England, has turned our attention to practical solutions for our problems of infant and child mortality.

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* "Infant Welfare Work in Europe," by Nettie McGill, Community Child-welfare Series No. 1, Bureau Pub. No. 76, p. 21. Children's Bureau, U. S. Department of Labor, Washingtion, D. C., 1921.

Contribution of Our Universities.—Our Universities and Colleges for some time have attacked the problems of infancy and childhood from an academic standpoint, and have laid down certain fundamental guiding principles in child study; in psychology, in economics and in dietetics. The application of these principles has fallen largely to educated mothers, teachers, social workers and socio-medical pioneers. The questions involved are no longer of purely academic interest as to *what* we shall do for the children of our generation, but are of more immediate practical import as to *how* we shall marshal our medical and social forces to guarantee a future generation able to resist the disintegrating forces and to conserve elements of strength for the reconstruction period.

Practical Activities Stimulated by Universities.—The practical interest which our universities have developed in child welfare has grown out of a consciousness that mere academic regard for the underlying principles is productive of little enthusiasm in the student unless he comes into direct contact with the living problems. The paths to the larger interests in the problems of infant and child life were blazed by the patient, and ofttimes unappreciated, efforts of our University professors who have seen the wider social horizon. The first objective responses to their inspiration were met in university settlements; in experimental child study schools; in kindergartens and in juvenile courts. The milk stations and infant welfare centers were of somewhat later growth, but their function was immediately recognized as an important one in the great campaign against infant mortality. The study of tuberculosis in its various aspects had brought home to us that there were other than strictly medical phases in the problem of disease. Diseases such as tuberculosis and syphilis came to be regarded as largely social, to be met by social readjustments as well as by clinical medicine. To meet the pressing needs of the enlarging dispensary problem, social service departments connected with hospitals arose.

Responsibility of the Medical School.*—The modern Medical School has had its share in the shaping of our ideas regarding the social as well as the medical treatment of children. This is aptly illustrated in the development of the Pediatric Department, as differentiated from General Medicine, in some of the leading institutions. In these places the pediatric department has often been the pioneer in socio-medical work. Lucas in writing upon the subject of the socio-medical development in our medical schools has said that: "This growth has been rapid and gratifying to all those who have had an opportunity to follow the development in its entirety, and such growth should set the standard for the development of the other legitimate fields in pediatrics, for it is only by the unification of laboratory, hospital and social factors that any of the problems of child welfare can be worked out satisfactorily. The medical schools, even in this field, have differed in the emphasis put on these three lines of development. In some schools the purely teaching phase has predominated, while others have dwelt largely on research work in infant metabolism, and again, the social work has lagged far behind. But from the field of pediatrics as a whole, certain high standards have emerged along all lines, and not the least of these is the child welfare social work as it is being carried on by the New York Milk Committee and by the pediatric departments of Harvard and Western Reserve Universities."*

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* Considerable material in the following sections has been derived from a "Thesis: The Problems Presented to a Children's Department of a University Hospital in Meeting the Medical-social Needs of a Cosmopolitan City," by Richard A. Bolt, presented to the University of California, May, 1917.

Social Pediatrics.—Since this was written considerable advance has been made in a number of our medical schools in providing certain work in "social pediatrics." The American Pediatric Society has taken cognizance of this work and has provided as a part of its annual program a section on "Child Welfare." A Committee appointed by this Society also drew up an outline of what it considered desirable in the teaching of medical students in Child Hygiene. Dr. Henry L. K. Shaw has presented a very helpful syllabus of a course of instruction in social medicine which is being carried out at Albany Medical College.† If the medical man of the future is to solve intelligently the problems connected with infant mortality he must, it would seem, receive thorough instruction in the fundamentals of child welfare during his medical course. The following seem essential in infant and child welfare work:

An Ideal Scheme for a Social Pediatric Course.‡—1. A thorough course in pediatrics, laying stress on the fundamentals of infant hygiene and infant feeding.

2. Thorough instruction in modern pediatric methods with actual experience in a babies' dipensary for sick children and an infant welfare center for well ones.

3. A good working knowledge of obstetrics, especially in its relation to the nursing and social needs of the community.

4. Experience in a maternity and prenatal service.

5. A knowledge of the important causes of infant mortality and the most approved methods for its prevention.

6. Familiarity with the general methods of all social agencies working in the community.

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* Lucas, William Palmer, Some Further Developments in a Pediatric Department of a University Medical School, Jour. A. M. A., lxvi (April 8, 1916), p. 1075.

† Shaw, H. L. K., Social Pediatrics, Jour. A. M. A., lxxiv (May 1, 1920), pp. 1275–76.

‡ Bolt, Richard A., The Education of the Medical Student in His Relation to Child Welfare. Reprint from "Canadian Medical Week" (1917).

Coördination Needed.—There is another very important phase of the child welfare program. Parallel with the work in our universities and colleges to solve the child hygiene problems, a considerable amount of extra mural effort has been expended by philanthropic individuals and social organizations. Their humanitarian impulses to "help the babies" were at first largely sentimental, and simply offered immediate and temporary balm for the crying needs of the child. Charitable effort was directed to "saving the child." The complex causes of infant mortality were at first dimly glimpsed. The intricate problems of infant mortality, of social and economic maladjustments, of mental deficiencies slowly came into view. Practical workers in the social field who came hard up against the every-day problems of childhood were eventually brought face to face with buoyant, enthusiastic students fresh from the sociology and economic classes who were anxious to try out the social and economic theories imbibed there. This give and take produced more hopeful, resourceful workers. It further stimulated the universities to provide facilities for first hand training of its students along socio-medical lines. This is distinctly reflected, during the past decade, in the active growth of child welfare in coöperation with the various medical and social agencies of our cosmopolitan centers.

Relation of Private Initiative to Public Health Work.—While pioneer work in child welfare has been done almost entirely by private agencies or individuals—or at most by those of a semi-private nature financed by private subscription—it did not take long for the public to realize that much of the work done by child welfare organizations was really public health work. Little by little certain parts of the child welfare program have been taken over by the municipal health departments in our more progressive cities. The logical outcome is to have this public health work for children taken over and directed by the health authorities of the community. Thus far comparatively few health departments are in a position to carry on successfully such work. Too often the departments are hampered by politics and poorly paid officials. The budgets for most health departments are woefully inadequate to meet the needs, and the money allotted for child welfare is infinitesimal compared to that devoted to other municipal purposes. The voluntary, private organizations must for some time back up the work attempted by the local health officials, and give a stimulus to it.

Growth of State and City Bureaus of Child Hygiene.[*]— A development of considerable promise has been the organization of a large number of State Bureaus of Child Hygiene. Child hygiene being a very important part of general health should receive adequate recognition and support from the State authorities. The first State Child Hygiene Division was established in Louisiana in 1912, although it should be borne in mind that several cities had established Bureaus of

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[*] Rude, Anna E., Status of State Bureaus of Child Hygiene, American Journal of Public Health (Oct., 1920), 10, pp. 772–779.

Child Hygiene prior to that time. New York City established its Bureau in 1908. In the early years the growth of divisions of child hygiene in state health departments was very slow, only eight being established in the years 1912–1917. In the period 1918–1920, due to the stimulus of the War and the nation wide interest aroused by the Children's Year, 23 state divisions or bureaus of child hygiene were set up. Since then five more have been established, making a grand total of 36 states now organized to carry on child hygiene. Besides the organization of bureaus of child hygiene, a considerable amount of study has been put upon child welfare problems in the various states by so called Children's Code Commissions,* appointed in most instances by the Governors to study the conditions and make recommendations as to necessary legislation. While it is true that the greater part of the work of these Commissions concerned itself with dependent, delinquent and defective children and institutional care, some of the states have had a careful survey made of the health conditions. The National Child Health Council recognizing the importance of the work of these Commissions appointed an advisory committee to study the work of the Child Welfare Commissions and suggest minimum requirements. A great deal remains to be done in bringing the child welfare work into conformity with what we now know to be suitable standards. A large number of national organizations are attempting to carry on educational publicity along various lines of child welfare and are stimulating local efforts to meet the problems.†

Universities Must Provide for Training in Child Welfare.—With all this broad development of child welfare the University must reckon. The training necessary to equip physicians, nurses and directors for child hygiene bureaus is properly the function of the University. The Medical School through its Department of Pediatrics should be alive to the responsibilities involved in the training of socially minded men and women. Nowhere has the social change in medicine been more marked than in the pediatric departments of our large university medical schools. In sketching an ideal scheme for future public health work, bearing in mind especially the child hygiene side, Gerstenberger, one of the pioneers in this field, says that one of its fundamental characteristics would be "the supervision and constant education of this physician (the properly trained and supervised district physician) by the heads of the various university departments in medicine, sociology and nursing, or by members of their staffs appointed by them. I choose the University as the guiding and supervising institution for two reasons:

"1. Because with our present ever-changing form of government, the University is the only place where we can hope for stability, conservatism, permanency and ideals. This does not mean, of course, that stability, conservatism, permanency and ideals are always found at the University, but in our country they are most likely and most frequently to be found there rather than anywhere else.

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* State Commissions for the Study and Revision of Child Welfare Laws, Children's Bureau, U. S. Department of Labor, Publication No. 71 (1921).

† See Digest of Programs of National Organizations Carrying on Some Phase of Child Welfare. Published by the American Child Hygiene Association, 1921.

"2. Because the University in order to best train men and women for such work needs direct access to the practical public health work."*

A Modern Pediatric Department.—The function of a modern pediatric department of a university medical school is, therefore, no longer merely to instruct how to examine and prescribe for sick babies, nor to utilize these as so much "clinical material" for the instruction of medical students. The use of the babies for instructive purposes must of course not be overlooked, but it involves a wider social significance than formerly believed. The educational program must take into consideration the well baby, its mother, the home and its social and economic circumstances. These will throw valuable side-lights on the strictly medical problems and greatly assist in successfully carrying out their solution. The relation of pediatrics to public health will inevitably receive more attention. "Public Health work for children," said Dr. S. Josephine Baker, "has steadily and progressively deviated from our preconceived ideas of the relation of health boards to the community. It is an expression of the social conscience of the people together with an enlightened desire on the part of the health authorities to direct and educate rather than to correct and punish."

Local Responsibility for Infant Welfare.—Two decades of education and patient endeavor on the part of voluntary organizations and philanthropic individuals, with a wealth of experience in the various phases of infant welfare work, have thoroughly demonstrated that the local community is largely responsible for the protection and conservation of its child life. It is now generally admitted that the city or town not only has the right to throw about motherhood and childhood every possible safeguard, but that it has become its sacred duty to do so. The Nation itself has at last awakened to its responsibility and after prolonged hearings before Senate and House Committees and consideration before both branches of Congress a Bill for the "Public Protection of Maternity and Infancy" has finally passed Congress. This Bill "was built upon the general plan of aid from the National Government to the States for the purpose of stimulating the States to greater activity in these regards and for the purpose as well of aiding them by contributions from the General Government."†

Tangible Results.—The value of consultations for nurslings, milk stations and infant welfare centers, first demonstrated in France, England and Germany, has also been amply shown in the United

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* Gerstenberger, H. J., The Question of Smaller Nursing Districts for All Kinds of Public Health Work versus Larger Districts for Specialized Work, Proc. Amer. Assoc. for Study and Prevent. Inf. Mort. (1915), p. 331.

† Towner, Horace M., Statement before the Committee on Interstate and Foreign Commerce of the House of Representatives at Hearings on the Sheppard-Towner Bill for the Public Protection of Maternity and Infancy, Washington, D. C. (Dec. 20, 1920).

States. They were the forerunners in the great campaign against infant mortality in its broadest sense. A study of the infant mortality rates in those cities and towns where intensive infant hygiene work has been carried on reveals a progressively declining rate during the past decade. In those places where painstaking records have been kept a comparison of the general infant mortality rate with that of babies coming directly under the supervision of welfare centers shows that clean, pasteurized milk, expert directions as to feeding and hygiene and follow-up in the homes by trained nurses have had a decided effect in reducing the mortalities of infancy. A similar result has followed in lessened maternal and neonatal mortality where well conceived prenatal work has been carried out.

Better Birth Registration Needed.—Although it is quite impossible in many places, on account of deficiency in birth registration, to state exactly what the reduction has been, it is safe to say that it has been quite substantial. The reduction in the actual number of deaths of infants, despite the great increase in population and number of births, shows that our contention is sound. If we had complete birth registration in this country it would be immediately reflected in the infant mortality rates, and we would be able to know the exact trend of infant mortality and measure the results of our efforts. This desideratum can only be obtained by encouraging a keener civic conscience on the part of physicians and midwives, stimulated by an informed public opinion, and enforced by a fearless wielding of the big stick on the part of the State and municipal authorities.

Development of Infant Welfare Centers.*—While "milk stations" and "consultations" blazed the trail to better efforts, we must now look upon them as simply steps in the gradual evolution of infant welfare centers, known variously as "Baby Health Conferences," "Infant Welfare Stations," "Prophylactic Dispensaries" (Cleveland), etc. The fundamental principles, which were laid down early guiding all these endeavors have remained the same, namely, getting the mother to bring the baby to the consultation as early as possible; the encouragement of breast feeding; thorough physical examination and periodic weighing of the baby; prescription for modification of milk in those cases unable to breast feed; general hygienic directions; follow-up and instruction by nurses in the homes. While, as is said, these have remained practically the same our field of public health service has greatly broadened and we have been led step by step to integrate the infant welfare work of the community with the enlarged public health work.

Infant Welfare Center as Part of Health Center.—To meet the insistent needs of a city-wide public health service for the children—especially for those of tender years—it seems best to group our work into so-called infant welfare centers as integral parts of health centers, to become completely coördinated with all the public health activities of a health district. This, of course, still leaves ample room for voluntary efforts in supplementing the work of the official agencies and in carrying forward new developments and experimental work.

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* See Bolt, Richard A., The Development of Infant Welfare Centers, The Cleveland Medical Journal, xvi (Feb., 1918), p. 69. Also Gibbon, I. G., Infant Welfare Centers, The National League for Physical Education and Improvement, Tavistock Square, W. C. London (1913).

Infant Welfare Center a Health Educational Center.—We must, henceforth, regard the Infant Welfare Center as the health educational center of the district in all that pertains to the welfare of the child. Here not only the mothers with their babies will meet the regular physician and nurses in attendance, but groups of medical students and pupil nurses will gather for instruction in the best methods of infant hygiene. We may also expect the extension of this work in postgraduate courses for physicians in the neighboring towns who care to brace up upon their infant feeding and methods of infant care. To this center the private physicians of the district may confidently come for the closest possible coöperation. They should be made to feel that here exists an opportunity for friendly consultation and study of the most approved methods of normal infant feeding. Much of the routine and detail work for well babies which a busy general practitioner does not care to carry out in his own office may be done in the center if he so desires. The "charity cases" may readily be taken off his hands. A cordial understanding between the private physician and the public health nurses in his district will do much to make his practice more effective.

Primary Function of Infant Welfare Center.—The infant welfare center should provide for the education of the mothers in the care, feeding and general hygiene of all normal infants irrespective of condition at birth or economic circumstances of parents. This is as much a part of the public function as public baths, public playgrounds, libraries or schools. It is often found that the "well-to-do and ignorant" are as much in need of instruction regarding the proper care of babies as the "poor but respectable." Instruction by properly trained and qualified public health nurses in the homes of the babies has been proved to be one of the most important, if not the most important, factor in keeping the babies well and in getting the mothers to take the baby to a physician or dispensary when the first signs of illness arise. Careful follow-up of all infants coming to an infant welfare center is absolutely essential to secure satisfactory results.

A Diagnostic Center.—Where the infant welfare center is a definite part of a district health center it is well to consider the advisability of utilizing it as a diagnostic clinic, as well as a clearing house for all normal well babies. This advance would afford the private physicians in the district an opportunity to avail themselves of expert diagnosis, fortified by laboratory tests, in all cases where it is difficult to carry out all the details in their own offices, or where the economic circumstances of the patients will not justify the extra time of a busy practitioner. While this extension of service would necessitate very careful adjustment in regard to private physicians, and a full understanding of just what the center may be expected to do, it is felt that the community, and reflexly the physician, will eventually be benefited.

The Normal Baby.—We should never lose sight of the fact that all infant welfare centers are primarily intended for the normal, well babies so that the mothers may receive proper instruction and advice in infant hygiene and that the course of development of the baby may be readily followed. This, of course, presupposes that the physicians and nurses in the centers are thoroughly familiar with what constitutes a normal baby, an expectation which is not always borne out in practice. Slight departures from the normal and mild gastro-intestinal disorders are often overlooked by the parents or glossed over by their family physician. In commenting upon this matter Grulee says: "The tendency to disregard slight gastro-intestinal symptoms is so widespread that one feels helpless in endeavoring to caution even the profession in this regard, but the recognition and proper treatment of slight gastro-intestinal disturbance is of much more importance than the ability to treat more severe conditions when they arise."*

Early Care.—These considerations have forced us to the conclusion that it is perfectly right and proper to encourage the mothers of all infants to bring them as early as possible to one of the infant welfare centers for a thorough preliminary examination and careful instructions in normal feeding, impressing the necessity of breast feeding and the hygiene of infancy. In England by means of the Notification of Births Act it has been possible for the Health Visitors to get into touch with the babies very early and thus give valuable advice to the mothers before they were able to get out to the centers. This advice is especially valuable in regard to the technique of breast feeding and simple hygiene of early infancy. Where the cases are in good financial circumstances and in the hands of capable physicians, a first call is sometimes all that is needed, and the mother is urged to consult her physician from time to time. It should be distinctly understood, and this must be made very plain to the mothers, that if the parents can afford a private physician the baby will be immediately referred out to him should any abnormality develop or should it become acutely ill. If parents cannot afford a private physician, and this will often have to be determined by a searching social investigation, the baby should be referred to a babies' dispensary and hospital or some other suitable institution in the community for sick babies.

Coöperation With Physicians.—The infant welfare centers stand in a position to coöperate with private physicians and render them all possible assistance in the way of sending incipient disorders to them, but have no intention of supplanting the physician in any way in his private work for sick babies. As a matter of fact, in those communities where well coördinated work for child welfare has been carried out the physicians in the community qualified to give an unbiased opinion have stated that such work has made their practice among children more satisfactory. The physicians and nurses connected with infant welfare centers should be especially trained for such work, and should receive compensation for their services, so as to command full respect from their professional associates not employed in such work. The nurses and physicians in the centers should seek to render every possible service consistent with their public responsibilities to physicians in their districts and should fully explain the purposes and methods of their work to the private physician.

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* Grulee, Clifford G., "Infant Feeding," 3d Edit. (1917), p. 67. See Chap. VI, "Attributes of the Normal Child."

The most delicate matter with which workers in infant welfare centers have to deal is in recommending some physician to the mother when she requests it. Many of the mothers going to centers have no regular family physician. In the poorer districts, moving about from place to place is quite common. In this age of wide social contacts no physician can consider that he has a mortgage on the family practice in any community, especially where he is called in but casually and sees the patient but once or twice. We must admit from sad experience that a considerable number of medical men have not had the training nor the experience to fit them to meet the emergencies of the sick infant, especially when it comes to difficult feeding cases. Certainly it is a grave responsibility for us to commit a baby into such hands. We must do either one of two things: Say to the mother that she must choose her own physician without any indication from us as to those of ability and experience in infant feeding, or mention to her a group of reliable medical men in her district whom we know to be competent to handle sick babies and difficult feeding cases. This matter may be worked out practically by bringing it before the local medical society and finding out what men are willing, and have had special training, to take these cases. In the larger centers it will not be difficult to work through a medical society but difficulty may arise in the smaller towns where every doctor on "Main Street" considers himself capable of taking care of any kind of a case. It should be borne in mind that the physicians engaged at the infant welfare centers are usually only on part time. It would be ideal if we could have full-time well trained men for this work, but this is out of the question in most places at present. Only under very exceptional circumstances therefore should any physician working at one of the centers be recommended to the mother in his capacity as a private physician. In the development of infant welfare centers it has been found wise never to *single out* a private physician either to recommend or condemn him. Should the mother give the name of her family physician we are then in a position to get into touch with him and explain to him what the center stands for.

Functions of an Infant Welfare Center.—Summing up the functions of an infant welfare center in a child welfare scheme we may say:

1. It should be open to all mothers and well babies in the district regardless of economic circumstances.
2. A thorough preliminary physical examination of every baby upon its entrance to the center by a well qualified physician.

3. Periodic consultations and weighing of all babies with careful records.

4. Instruction and advice to all mothers in the care, feeding and general hygiene of infancy.

5. Promotion of breast feeding. Where this fails, suitable instructions for the modification of cow's milk.

6. No special milks or formulæ designed for ill babies to be ordered from these centers. All babies needing such milk should be referred to a private physician or a babies' dispensary and hospital for examination and treatment.

7. When a baby under the observation of one of the infant welfare centers develops an abnormal condition or an acute illness, and its parents are in good economic circumstances, it should be referred immediately to a private physician. If unable to afford a private physician, it should be referred to a babies' dispensary and hospital or other institution where it can be suitably looked after. The nurse from the center should get in touch with the private physician or institution and give a history of the case to them.

In the more highly developed centers connected with dispensaries and hospitals the following may also be provided:

1. Smallpox vaccination for all babies whose parents cannot afford a private physician.

2. Securing of blood for Wassermann reactions in all babies sent to boarding homes, or for adoption, and for others where a diagnosis of syphilis is desirable.

3. von Pirquet test on babies from tuberculous homes and in others suspected of tuberculosis.

4. Schick test for babies exposed to diphtheria, where the question of administering immunizing doses of toxin-antitoxin comes up. The ideal method would be to immunize all children between the ages of six months and six years.

5. Taking of nose and throat cultures from all suspicious cases.

6. Taking of smears from inflamed eyes showing secretion.

7. Taking of vaginal smears in all babies sent to boarding homes or to a hospital or out-door ward.

Materials for this diagnostic work should be furnished by the local Department of Health, and the examination of the blood, smears, etc., carried out in the Department Laboratories free of charge to the physicians sending in cases or to the parents who bring the babies.

Future Developments of Infant Welfare Work.—We may reasonably expect that future developments in the saving of infant lives and promoting their welfare will be along the following lines:

1. The recognition of infant welfare work as an integral part of a full rounded maternity and child hygiene program.

2. Better training of physicians, nurses and social workers in socio-medical work for infants and older children.

3. The organization of community forces to cope with the untoward conditions which contribute to infant morbidity and mortality.

4. The prevention of prenatal and neonatal mortality by adequate prenatal care, more skilled obstetrics and better service in maternity hospitals and convalescent homes for mothers.

5. The registration and strict supervision of *all* midwives.

6. The pasteurization or sterilization of *all* milk given to babies.

7. Widespread revival of interest in breast feeding and its promotion.

8. Prevention of whooping-cough and measles in infancy.

9. After six months of age, the immunization against diphtheria by use of toxin-antitoxin.

10. A revival of the practice of vaccination against smallpox for *all* infants.

11. An extension to the rural districts of the lessons learned in the cities.

12. The better organization of state and municipal bureaus of child hygiene to carry on a widespread popular education regarding infant care.

13. Larger interest of the Federal Government in stimulating local endeavors by the granting of State subsidies.

When all these measures are under way we may well expect an infant mortality rate of less than 50 throughout the country and a more healthy and resistant childhood at all ages above one year.

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CHAPTER XIII

HISTORY TAKING AND THE PHYSICAL EXAMINATION OF INFANTS AND CHILDREN

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THE CLINICAL EXAMINATION OF CHILDREN

The modern pediatrician is fundamentally different in training, in ideals and practice, from the old-fashioned doctor who was "good with children." The empiric doctrines of 40 years ago have been modified and changed by experimental research and conclusions based on facts. Laboratories of chemistry, physics, bacteriology, physiology and pathology have furnished the physician with new means to study the various problems of disease, and the studies of Virchow, Pasteur, Koch and the other earlier investigators have been elaborated to such an extent that there is a constant and progressive flow of the results of research.

New classifications of symptoms and diseases have been evolved, based not only on bedside observations but, as well, on untiring investigation in the laboratory. Chemical and physical methods, colorimetric apparatus and serological tests, have been introduced in the study of the various fluids of the body. The specialist in diseases of the heart feels that he cannot do complete work without an electrocardiograph and orthodiagraph at his disposal. New functional tests are reported at frequent intervals in current literature.

The diagnostician of today need not necessarily be a practical laboratory technician, but he must have the ability to interpret the various tests, and he can keep pace with modern diagnostic progress only by constant study.

Czerny and Finkelstein stand out as leaders in the modern European school of infantile nutrition. Czerny studied infants at the bedside and gave us a classification based on a chemical etiology; while Finkelstein introduced a most practical classification based on symptomatology, recognizing, however, the importance of making an etiologic diagnosis. The work of these two men alone has inspired countless others to work with them to carry their ideas to completion. At no time have so many advances been made in scientific pediatrics as in the last 15 years.

The day is past when the physician can travel from house to house with no instruments except his stethoscope and thermometer. If he attempts, however, to practice with only a test-tube and a guinea-pig his results will be even more unsatisfactory. The clinician must combine the clinic with the laboratory. The keenness of the old-time observer should be emulated by the modern physician, and in this the physical diagnosis is of prime importance; the laboratory examination must be accepted for what it is worth both for its positive and negative values. The scientific physician collects his history data systematically and thoroughly, examines his patient carefully, calls upon the laboratory for all necessary aid and correlates all the facts he has gathered. He is then prepared to arrive at a solid conclusion, based on his experience, his keenness of observation and his good sane judgment. The student, or interne, should be taught to think along these lines. He should be instructed in careful history writing, the technic of examining the patient, whether infant, child or adult, and to check up the findings of the laboratory.

The teacher of the future will be obliged to devote his entire time to teaching and research. Many medical schools today demand that professors and assistants refrain from private practice so that they may have the time to study the cases in the hospital and out-patient department, in all phases and from all angles.

But we may ask whether men who have never had successful practices can teach the student to diagnose and treat the sick. The best answer is that the spirit for investigation and study among the students in such schools is far greater than in those in which the main purpose is to absorb a collection of facts from a textbook or a hurriedly prepared lecture. Thoroughness in the examination of cases is the keynote.

The busy doctor may question whether he can keep up with all the new investigations. He travels from house to house all day long and comes home late at night too tired to read and unable to concentrate. He recognizes his deficiencies. But what can he do? He has no time and is unprepared to do laboratory work and his practice does not afford him sufficient income to hire a skilled assistant. His fees are small and he feels that he cannot demand charges for special work. But when the patient dismisses the busy doctor and consults the specialist, who studies every detail of his ailment, he pays the price cheerfully and leaves with satisfaction. He will never forgive the physician who missed the diagnosis because he was too busy or too poorly equipped to examine, for instance, the blood.

Specialization is by no means essential to good medical practice. In many small communities it is out of the question, but careful history taking and thorough examination will eliminate many diagnostic mistakes. Errors of commission depend to a large extent upon mistaken judgment and lack of experience, but errors of omission are primarily due to carelessness.

Within recent years numerous medical groups have been organized, especially in the smaller cities throughout the middle west and western states. Each man in the group has his own particular specialty and there is a central clinical laboratory, *x*-ray department and business office. Routine and special laboratory work is done on each patient, cases are referred from one department to another and the records are systematized and well kept up. The patient is studied from every angle and receives as careful an examination as the charity patient in a charity hospital. It has long been recognized that the millionaire and the pauper have the advantage from a medical standpoint. The great middle class has not yet come into its own.

The Case History.—The tendency to elicit careful histories and to formulate the acquired facts on suitable charts is modern. It is only within recent years that the student in medical schools and hospitals has been given adequate instruction in systematic history taking. The older clinician was content with a card upon which a few points were carelessly jotted down. The modern student in the better schools and hospitals is compelled to write complete histories and to keep up his records with notations from day to day. His work is supervised and criticized and when he graduates he at least knows what a good history should contain, even though he does not carry out his teachings in his practice.

In taking the history of an adult the physician asks himself why the patient comes for examination. He may have a concrete ailment, but direct questioning leads to localization of the trouble and its intensity and under careful examination a complete history is ultimately obtained. In practice among children, however, different methods must be pursued. The baby cannot tell his own story, so the physician must depend upon observation and statements of the mother or the nurse. Even an older child, in the pre-school age, cannot give a reliable history. The element of fear or bashfulness in the presence of the doctor may dispel all symptoms from his memory. In these cases we must depend upon the facts as the child has disclosed them to the nurse or mother, and upon her intelligence in grouping the facts.

If we question the child concerning his pain, he may have difficulty in localizing it. A pain in the chest is commonly referred to the abdomen; a sore throat to the teeth or gums. The young child very often does not distinguish between pain and discomfort. A feeling of nausea may be interpreted by him as pain. An infant not infrequently indicates the location of pain by his movements. If his ear aches he may pull at the ear; if he has abdominal pain he may lie with his thighs flexed upon the abdomen. Even in such a case we must depend upon the mother's interpretation or upon our own observations and the symptoms are accordingly objective—not subjective.

Not rarely a child feigns illness. Many a headache that appears suddenly before school time disappears even more rapidly after the school bell has rung. Nevertheless, the hypochondriasis that is observed so frequently in adults, that leads us into wrong paths, is not so prominent a factor in practice among children. It is readily understood that subjective symptoms in early childhood are of minor importance. Indeed we may depend upon objective symptoms entirely or upon subjective symptoms as revealed by the parent.

The excited mother's story, with its distorted observations and flagrant exaggerations might tend to minimize in our minds the importance of the clinical history. She affirms and repeats again that the child eats absolutely nothing, but the well-fed appearance of the baby belies her statement. She maintains that the bowels are in perfect condition, but cross questioning reveals that the stools are too frequent, or even watery. Constipation may be the chief complaint, the reason for the baby's visit to us. We question her and learn that one or two well-digested and formed evacuations are in her mind insufficient. However, if we proceed with patience and endeavor not to excite still further an already excited mother and at the same time hold her to definite facts, a history can be procured that will aid us considerably and make the examination more easy.

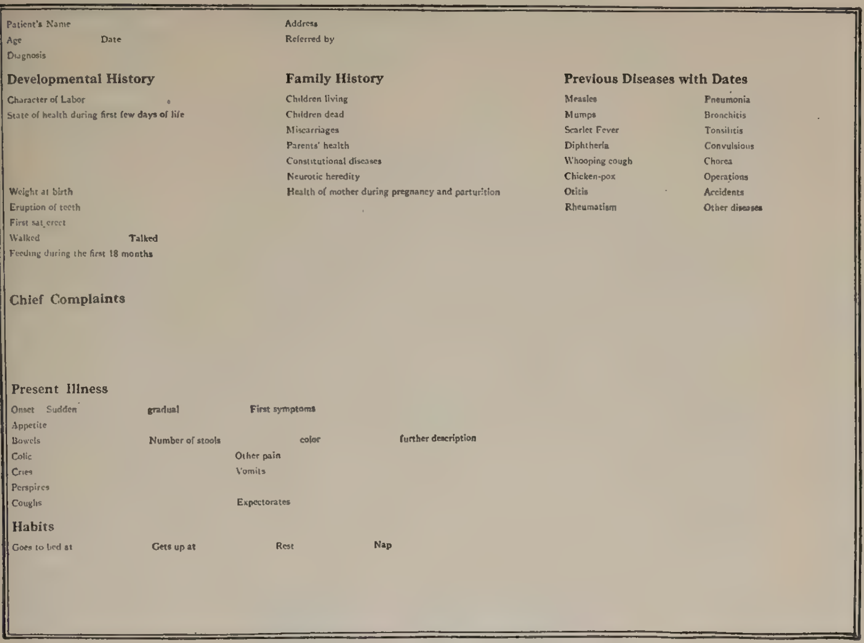
The mother or nurse should be allowed to tell the story in her own easier way and without interruption, so far as is possible. When she has completed her story we may then question her and eliminate unimportant facts from our minds, enlarging upon the important features, passed over by her as unessential.

Charts and Outlines for History Taking

Numerous methods of history charting have been recommended. Many clinicians prefer a blank card or folder containing no printed matter. A flexible outline is followed. Others adopt the opposite extreme and employ a chart filled with printed guides, which can be answered "yes" or "no" or merely checked. The first plan permits greater individuality and the avoidance of unnecessary detail to suit each case. The other method, while of great benefit for statistical purposes, has the tendency to produce stereotyped histories without logical sequence.

A chart which may be said to hold a middle position takes the form of a four-page folder, each page measuring 11½ inches wide and 8½ inches high, a convenient shape for filing. (See Figs. 21–24.) Page 1 covers the history in detail. The printed matter, as may be seen by the illustration, permits certain questions to be answered by a word or two, and ensures that the main points of the history shall be thoroughly covered. The physician has sufficient latitude to consider the individual history of each patient. Page 2 is devoted to the physical examination and requires no explanation. Page 3 takes up the special laboratory findings under which may be recorded the result of examination of the gastric contents, feces, sputum, pleural fluid, ascitic fluid, pericarditic fluid, spinal fluid, presence of pus, tuberculin tests, Shick test, and skin-protein reaction. Page 4 covers treatment and subsequent visits. If necessary a blank sheet may be inserted to cover further visits.

If the physician should wish to use his records for case reports or for statistical purposes, the supplementary pages may be used. (See Figs. 25, 26.) This consists of a sheet, printed on both sides, with



Patient's Name
Age Date
Diagnosis

Address
Referred by

Developmental History

Character of Labor
State of health during first few days of life
Weight at birth
Eruption of teeth
First sat erect
Walked Talked
Feeding during the first 18 months

Family History

Children living
Children dead
Miscarriages
Parents' health
Constitutional diseases
Neurotic heredity
Health of mother during pregnancy and parturition

Previous Diseases with Dates

Measles
Mumps
Scarlet Fever
Diphtheria
Whooping cough
Chicken-pox
Otitis
Rheumatism

Pneumonia
Bronchitis
Tonsilitis
Convulsions
Chorea
Operations
Accidents
Other diseases

Chief Complaints

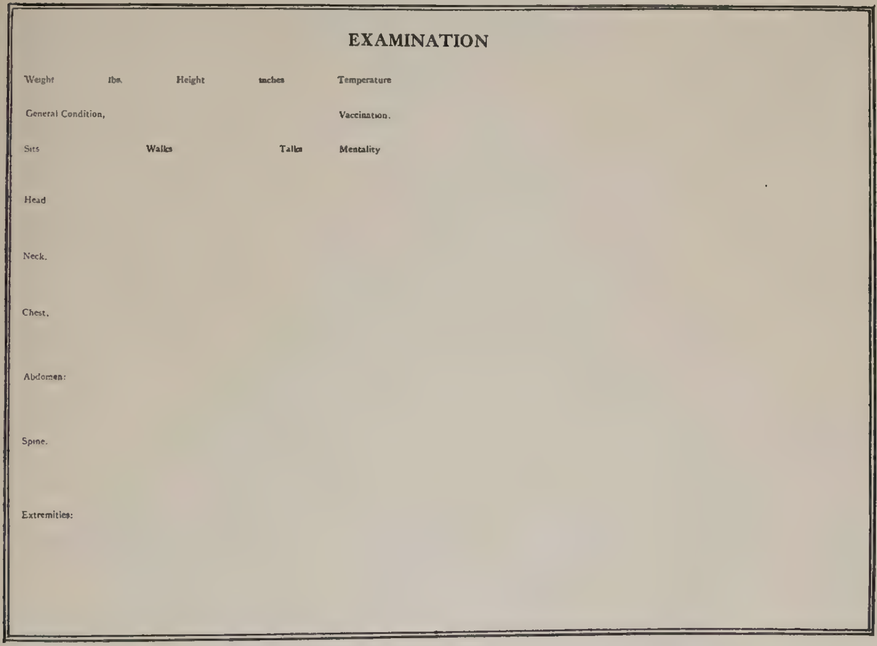
Present Illness

Onset Sudden gradual First symptoms
Appetite
Bowels Number of stools color further description
Colic Other pain
Cries Vomits
Perspires
Coughs Expectorates

Habits

Goes to bed at Gets up at Rest Nap

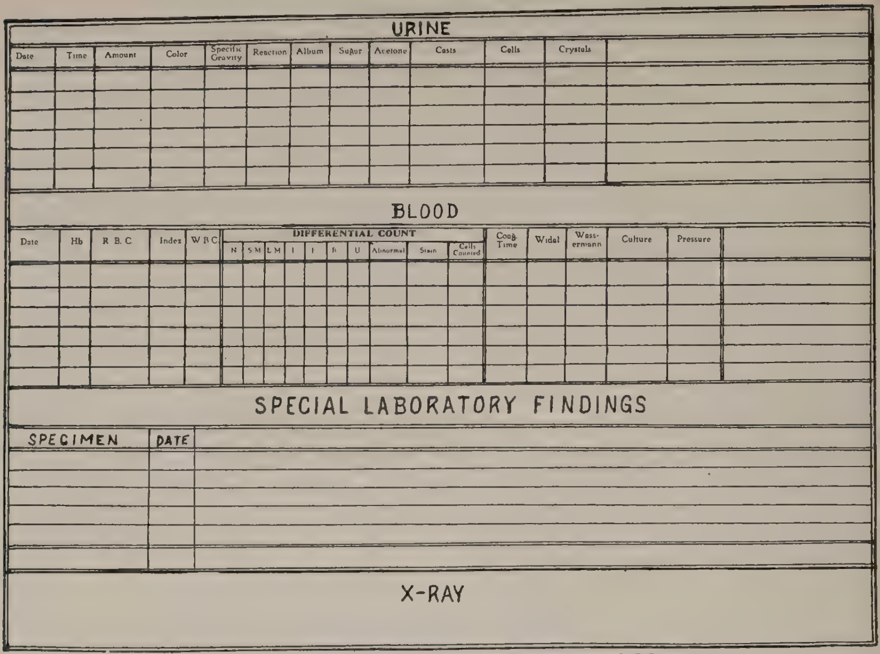
FIG. 21.—CHART I. Page 1 of history folder.



EXAMINATION

Weight lbs. Height inches Temperature
General Condition, Vaccination.
Sits Walks Talks Mentality
Head
Neck.
Chest.
Abdomen:
Spine.
Extremities:

FIG. 22.—CHART II. Page 2 of history folder.



URINE

Date	Time	Amount	Color	Specific Gravity	Reaction	Album	Sugar	Acetone	Casts	Cells	Crystals	

BLOOD

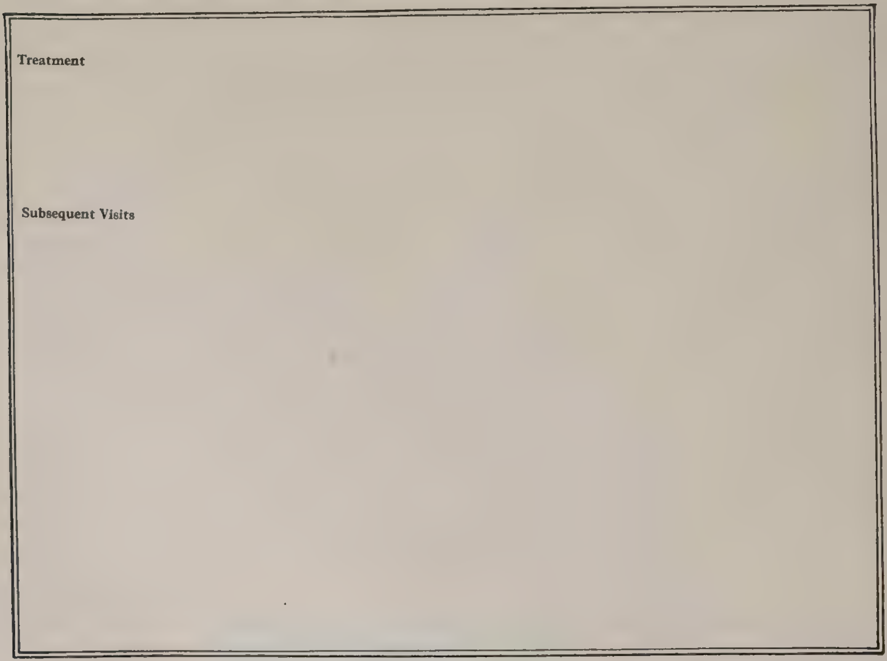
Date	Hb	R. B. C.	Index	W. B. C.	DIFFERENTIAL COUNT										Coag. Time	Widal	Wassermann	Culture	Pressure	
					N	S M	L M	T	E	B	U	Abnormal	Stain	Cells Counted						

SPECIAL LABORATORY FINDINGS

SPECIMEN	DATE	

X-RAY

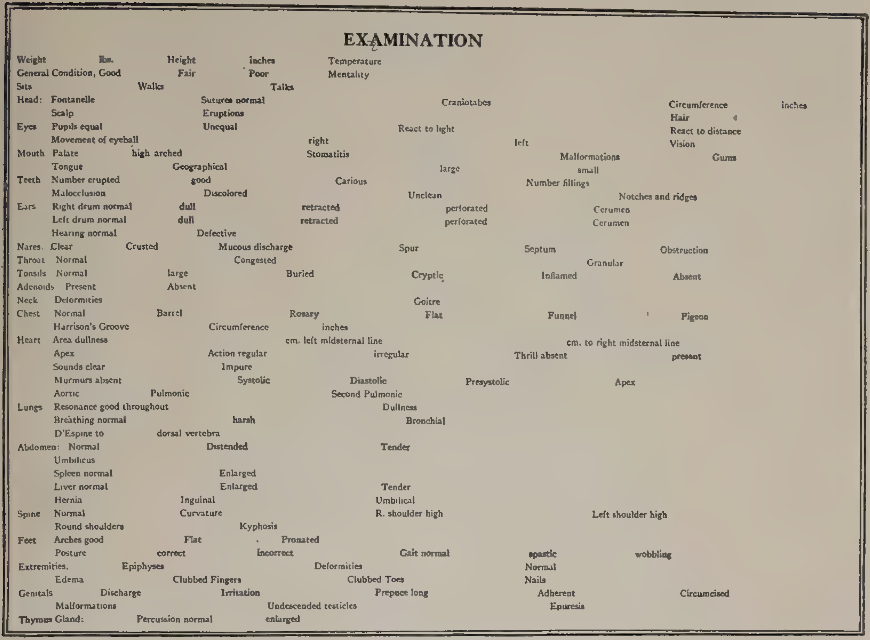
Fig. 23.—Chart III. Page 3 of history folder.



Treatment

Subsequent Visits

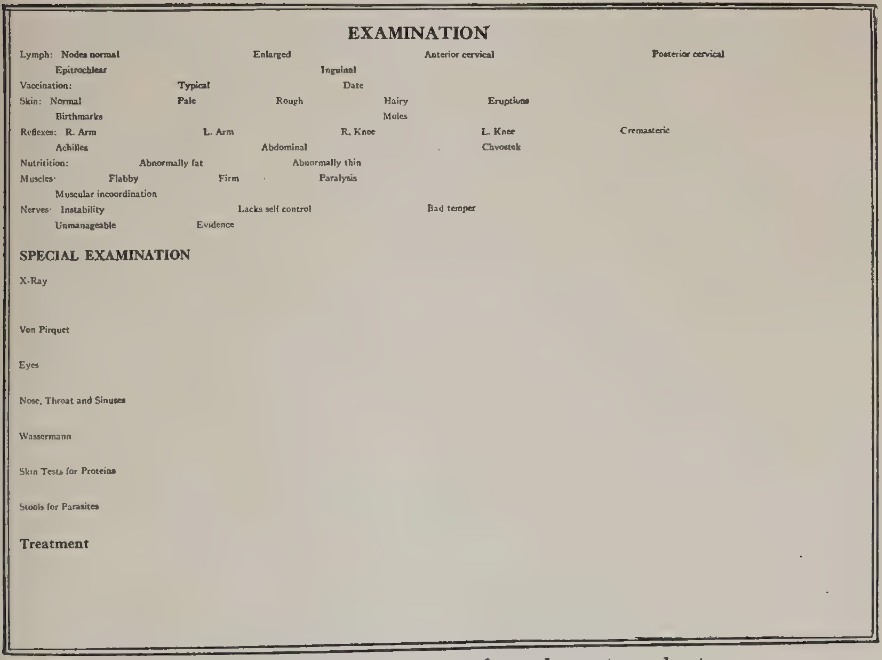
Fig. 24.—Chart IV. Page 4 of history folder.



EXAMINATION

Weight lbs. Height inches Temperature
General Condition, Good Fair Poor Mentality
Sits Walks Talks
Head: Fontanelle Sutures normal Craniotabes Circumference inches
Scalp Eruptions Hair
Eyes Pupils equal Unequal React to light React to distance
Movement of eyeball right left Vision
Mouth Palate high arched Stomatitis Malformations Gums
Tongue Geographical large small
Teeth Number erupted good Carious Number fillings
Malocclusion Discolored Unclean Notches and ridges
Ears Right drum normal dull retracted perforated Cerumen
Left drum normal dull retracted perforated Cerumen
Hearing normal Defective
Nares. Clear Crusted Mucous discharge Spur Septum Obstruction
Throat Normal Congested Granular
Tonsils Normal large Buried Cryptic Inflamed Absent
Adenoids Present Absent
Neck Deformities Goitre
Chest Normal Barrel Rosary Flat Funnel Pigeon
Harrison's Groove Circumference inches
Heart Area dullness cm. left midsternal line cm. to right midsternal line
Apex Action regular irregular Thrill absent present
Sounds clear Impure
Murmurs absent Systolic Diastolic Presystolic Apex
Aortic Pulmonic Second Pulmonic
Lungs Resonance good throughout Dullness
Breathing normal harsh Bronchial
D'Espine to dorsal vertebra
Abdomen: Normal Distended Tender
Umbilicus
Spleen normal Enlarged
Liver normal Enlarged Tender
Hernia Inguinal Umbilical
Spine Normal Curvature R. shoulder high Left shoulder high
Round shoulders Kyphosis
Feet Arches good Flat Pronated
Posture correct incorrect Gait normal spastic wobbling
Extremities. Epiphyses Deformities Normal
Edema Clubbed Fingers Clubbed Toes Nails
Genitals Discharge Irritation Prepuce long Adherent Circumcised
Malformations Undescended testicles Enuresis
Thymus Gland: Percussion normal enlarged

FIG. 25.—CHART V. Page 1 of supplementary sheet for statistical purposes. Not practical for office routine.



EXAMINATION

Lymph: Nodes normal Enlarged Anterior cervical Posterior cervical
Epitrochlear Inguinal
Vaccination: Typical Date
Skin: Normal Pale Rough Hairy Eruptions
Birthmarks Moles
Reflexes: R. Arm L. Arm R. Knee L. Knee Cremasteric
Achilles Abdominal Chvostek
Nutrition: Abnormally fat Abnormally thin
Muscles: Flabby Firm Paralysis
Muscular incoordination
Nerves: Instability Lacks self control Bad temper
Unmanageable Evidence

SPECIAL EXAMINATION

X-Ray

Von Pirquet

Eyes

Nose, Throat and Sinuses

Wassermann

Skin Tests for Proteins

Stools for Parasites

Treatment

FIG. 26.—CHART VI. Page 2 of supplementary sheet.

guides to cover all requirements. In the ordinary office routine they are objectionable because the important points do not stand out and the time consumed in recording the negative points is not compensated for by the results.

DETAILS

Present Trouble.—The date of onset, the first symptoms and the duration of the illness should be ascertained. Was the onset sudden and accompanied by high fever with chills or convulsions? Such an onset suggests at once a severe general infection—sepsis, pneumonia, acute meningitis, scarlatina, erysipelas, tonsilitis, pyelitis, otitis media or some other local suppurative process. Was the onset insidious? If so what prodromes were observed and in what order did the symptoms appear? Were they present constantly or intermittently?

After investigating the mother's story of the present trouble, the physician should elicit further facts by questions concerning the various systems of the body in some definite order. By such a procedure the entire symptomatology of the child can be covered without overlooking any vital features.

Previous Illness.—All previous diseases and injuries should be recorded. We should note the dates, the duration and the severity of the attacks. We should learn whether the child has exhibited any susceptibility to special disorders. Children are commonly prone to respiratory infections, colds and coughs, sore throat, earache and running ears, croup and asthma. We should also determine whether the tonsils and adenoids have been removed and what were the local and constitutional effects of the operation. Repeated attacks of respiratory infection may lead to rheumatism and endocarditis.

Gastro-intestinal disorders are of frequent occurrence and we question concerning repeated attacks of diarrhea, constipation, indigestion, vomiting spells, meteorism, and as to the condition and care of the teeth.

Many children show a predisposition to nervous manifestations. Such children may give a history of extreme restlessness, irritability, insomnia, lack of attention in school and at home, night terrors, convulsions, twitchings or tics, and even hysterical attacks.

The various acute illnesses and exanthemata should be recorded, with the dates of occurrence. Scarlet fever, measles, German measles, whooping cough, chickenpox, and mumps rarely occur more than once and oftentimes the knowledge of the previous occurrence of these diseases will, by exclusion, aid us in our diagnosis.

We should determine whether the child has been healthy and robust or weak, sickly and always complaining.

Family History.—The present health and past history of the parents should be ascertained first. If the parents are advanced in years their ages should be recorded. It is commonly believed that the offspring of such parents is likely to be weak or defective but the truth of this may be questioned.

The health of the mother during the puerperal and lactation periods is important, especially if the patient is an infant. The nursing mother who is again pregnant or who suffers from tuberculosis should not nurse her baby. Tuberculosis, syphilis, gout, diabetes, neuropathic and psycopathic manifestations, alcoholism in either parent, influence the child's health and even the mental state. Tuberculosis is practically never transmitted to the fetus, but a tuberculous parent, particularly a nursing mother, living in the same house is a source of danger to the child by direct contagion. Finesse and tact are required in questioning concerning syphilis and alcoholism, and the mother should be questioned directly only as a last resort.

At times the occurrence of an acute infection in a nursing mother may have marked influence on the child's existence. During lactation the susceptibility of the mother to whooping-cough is increased and new-born infants are also especially liable to such infection. The physician may be placed in the difficult quandary, whether or not to separate the child from its mother. This does not apply to all of the infectious diseases; it is rare indeed to observe scarlet fever or measles in a nursing infant.

Consanguinity in the parents predisposes the child to deafmutism and choroidal diseases. The effect of inbreeding upon the mentality of the infant depends undoubtedly upon the character of the parents. If any vices or defects predominate in the family these may be intensified in the offspring, but the same may apply equally to the good qualities and virtues.

The influence of race and nationality must be considered. Amaurotic family idiocy is confined practically to Jews. Negroes and Italians in this country are especially prone to rickets, whether breast or bottle fed.

The number of living children with their ages and state of health should be noted, the coexistence of any similar disease among the children, as well as the number of children dead and the cause of their death. The number of miscarriages, the cause and approximate date of occurrence should be ascertained. In a large proportion of cases this history may be the first clew that leads to the diagnosis of congenital syphilis. The general constitutional vigor of the parents and the presence of constitutional diatheses have their influence on the child's welfare. Brilliant mentality, neurotic derangements of all kinds, idiocy, insanity, epilepsy, diabetes, eczema, gout, abnormal stature and physical eccentricities and deformities may be traced to a surprising degree through an entire family tree.

Developmental History.—The character of the labor and the state of health in the first few days of life should be recorded. The weight at birth and the main points in the development, such as the time and course of the eruption of the teeth and the age at which the child sat up, walked, and talked are of importance for purposes of comparison with the normal. The feeding in the early months of life should be considered with the developmental history.

THE PHYSICAL EXAMINATION

The routine of the physical examination in infants and young children must of necessity differ greatly from that employed with older children and adults. In practice among adults the physician may follow a definite outline and complete his examination according to it, but young children differ so vastly in disposition that each child must be approached in a different way. Most children have a fear of the spoon or the tongue depressor. For this reason the examination of the throat should, as a rule, be deferred to the end of the examination. Auscultation of the lungs or palpation of the abdomen of a crying or frightened child is at times impossible, and these examinations should be made at any propitious moment when the child is quiet.

As a rule the best results are obtained when we approach the baby quietly, pay little attention to him, touch him as little as possible and avoid all sudden shocks.

General Inspection of the Body.—A general impression of the child's condition is obtained by the mind at a glance, perhaps subconsciously. We note the posture, the facial expression, the nutritional condition, the degree of weakness or strength, the mental and nervous condition, the character of the respiration and the condition of the skin.

Much may be learned from a study of the face and the position of the body of our little patient. Are the features pinched? Is the expression bright, or stupid and apathetic? Does the child lie peaceful and quiet or is he disturbed and restless or suffering from pain? At no time does a child show such fear as when the doctor enters the room, in some cases. This is usually due to faulty training. Many parents and even nurses employ the physician as a "bogy-man" to frighten the child when he has misbehaved. The results of such training are self-evident. At no time does a child show the results of home training and discipline more clearly than during the visit with the doctor.

The general examination requires only a few seconds and without touching the child. Before proceeding with the examination it is advisable to follow some definite routine of inspection. The child should be completely undressed. In young infants, especially boys, for our own protection the diaper should not be removed until necessary. We should try to make friends with the child before proceeding with the examination. Children are frightened by loud noises. We should not speak sharply or gruffly on entering the room. It is even better to ignore the child completely and confine our conversation to the mother until he has become accustomed to our presence. Some children may become interested in a watch or stethoscope and may play with it while we are noting the mother's history. Forsyth prescribes three precautions which should be observed: First, don't handle the child more than is absolutely necessary; second, don't impede the movements of his limbs or head; and, third, don't startle him by unexpected actions. If we begin our examination by sudden palpation with cold hands or the application of the cold stethoscope to the skin, the child naturally resents it. It should be kept in mind that success in the examination depends on the attitude of the child toward the physician. Crying, as a rule, interferes with auscultation and also with the abdominal examination. One occasionally encounters children suffering from surgical abdominal disease in which the diagnosis was made only after an anesthetic was given. For these reasons we must be most careful in our method of approach. Strict or definite rules cannot be laid down. We must learn from experience. Some men naturally repel children; others attract. The success in handling children depends on the physicians' patience, his understanding of children, and the lessons learned by experience.

At times, nothing the physician does will quiet the child. He resents every effort of the doctor to be friendly. In such cases the best results are obtained when the mother is directed to leave the room. Frequently the child cries for the mother and promises to be good if the mother is allowed to return. If the child cries when she returns the simple threat usually suffices to quiet him. Occasionally we meet a child who is unaffected by any measure except force. These children must be held tight and compelled to submit without more ado. The examination is thereby shortened and the child realizes that resistance is of no avail. Most clinicians agree that a spoiled child is better behaved when away from the mother's influence.

Position of the Child during Examination.—Young infants may be examined either on a solid table or on the mother's lap, preferably on their backs across the mother's knees. Older children should sit on the mother's lap in a position parallel with the mother's body. The mother should face the light, with the child facing forward, supporting the child's back against her breast and holding the arms at the elbows. The older child is less likely to cry when sitting upright than when lying down. Later, when the child's confidence is gained he may be induced to lie on the bed or table, if necessary, to complete the examination. The child is now ready for the remainder of the examination. As has been said before, if the child is crying or fighting, the examination cannot be made in as definite an order as in an adult.

We learn as much as we can by inspecting the body without touching the child. Much of this part of the examination is carried on without the child's knowledge. In the examination of children we must follow our own judgment. If the child is crying the physician must perform that part of the examination with which crying does not interfere. Throat or ear examination or taking of the temperature should, as a rule, be postponed until the end of the examination.

Detailed Inspection of the Body.—After the general impressions have been noted a more detailed inspection of the body must be made. We have observed the physiogmomy, the expression of the eyes, the color of the skin and lips, the general nutrition; the position assumed in bed, the mental state, the character of the sleep or of the restlessness, and the respiration. We then note the shape of the head, the signs of diseases of the upper respiratory tract, abnormal pulsation or visible glandular enlargement in the neck, the contour and movements of the thorax, the condition of the spine, the location and character of the apex beat, precordial bulging, rachitic deformities, distention or retraction of the abdomen and visible peristalsis. We should endeavor to learn all that we can in this way. The physician of the old school depended to a great extent upon his keen observation and cultivated his faculties to such a degree that brilliant diagnoses were made seemingly by intuition. Sad to say, in these days of laboratory supremacy, real clinical observers are few and far between.

Palpation.—Having thoroughly inspected the child we come now to the more direct methods. Before touching the child the hands should be warmed. The patient should lie flat on his back with the abdominal muscles relaxed. If an acute surgical condition is suspected an anesthetic should be administered if relaxation cannot be secured in any other way. The examiner should stand at the right side of the patient. Passing his flat right hand lightly over the abdomen he notes the presence or absence of local tenderness or muscular rigidity and resistance. Abdominal tumors or enlargement or misplacement of the spleen, kidneys and liver may be detected. Gradually the palpation should be deeper and the left hand may be placed against the loin to facilitate the examination. At times, if the child resists and cries, if the examining hand is held in position, satisfactory results may be obtained during the inspiratory phase of his crying.

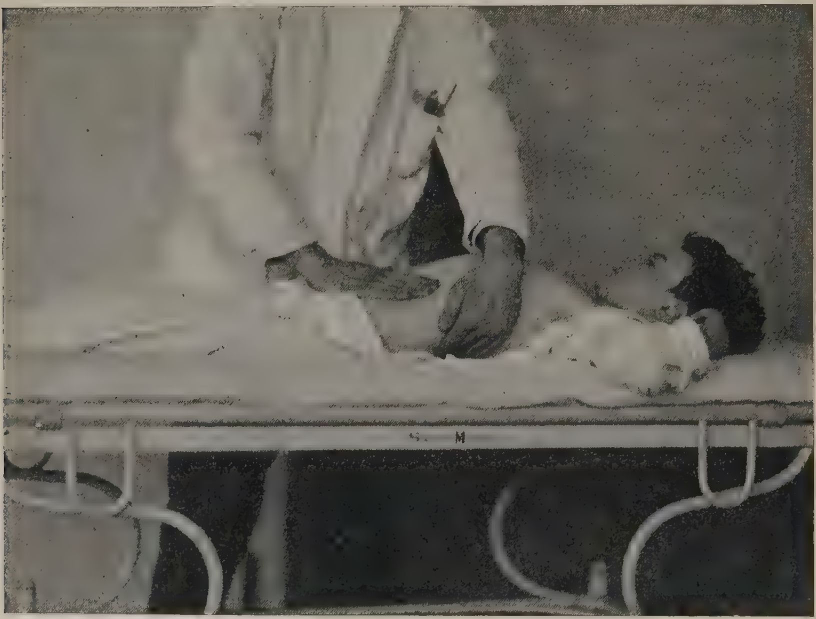


FIG. 27.—Position of child for palpation of abdomen. Method of palpating spleen.

The hand is then passed lightly over the thorax, noting the general contour of the chest and any deformities. The position of the apex beat, the presence or absence of cardiac thrill, bulging of the precordium or retraction of any region, and the character of the respiratory fremitus are observed. The head is next investigated, the condition of the fontanelles, the sutures, the general shape of the skull and whether cranio tabes exists. The examining hand then follows the neck to determine rigidity or relaxation of the muscles and the presence or absence of enlarged lymph nodes or abnormal pulsation. The spine should always be palpated. The hand then passes to the extremities, noting deformities, rigidity or flaccidity, painful, tender parts, the reflexes, the character of the radial pulse and finally attention is directed to the genitalia.

Percussion.—It is rather difficult to say whether auscultation or percussion should come first in the examination. Most clinicians believe that the stethoscope frightens the child more than the hands and in the first examination, at least, percuss before ausculting. Changes in the percussion note are more easily recognized in children than in adults because of the thinness of the thoracic walls. In a child, therefore, percussion should be very light and delicate. Deep percussion is apt to be misleading because of underlying structures. The finger, as a rule, is more satisfactory than a percussion hammer as it is not so likely to frighten the child and reveals as much as is learned by the hammer.

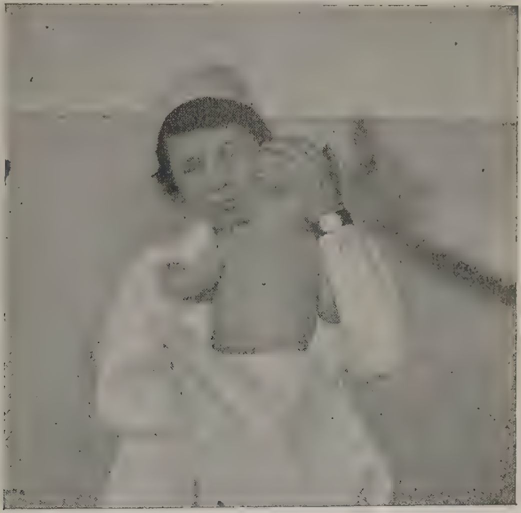


Fig. 28.—Method of holding infant for examination of the lungs posteriorly. In percussing the left chest the infant should be held over attendant's left shoulder; over right shoulder for right chest.

The child may be placed either in a sitting posture or flat on his back or face in bed. It is usually desirable to percuss the back first because the child is less frightened when not facing the physician.

Whatever the position we should be sure that the posture is straight. Twisting of the spine, as results from lying on the side or when the child is held over the mother's shoulder, causes contraction of the thoracic muscles and consequently impaired resonance. One may observe that when a child is held over the shoulder of the mother impaired resonance may be elicited. On transferring the child to the other shoulder the percussion note will become normal.

It is not easy to determine accurately the borders of the lungs and the degree of their mobility. The borders are higher in children than in adults. In infants the normal percussion note is more resonant than in adults and may even approach a tympanitic quality because of the thin chest walls and the relatively large bronchi. At the left base the note may be tympanitic because of the proximity of the stomach. In older children a slight hyporesonance may be noticed near the lower border of the left clavicle.

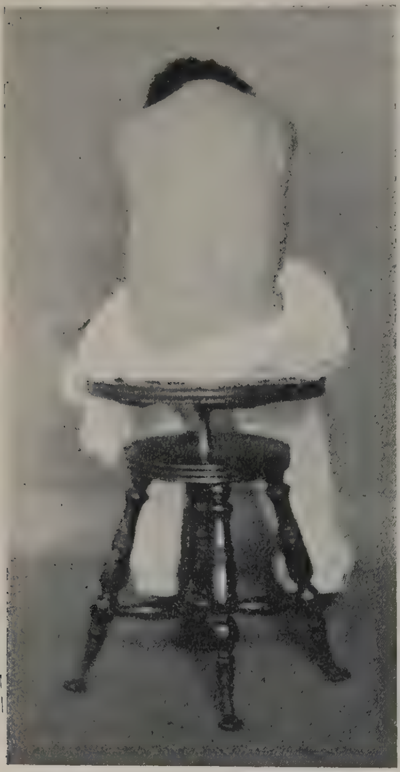


Fig. 29.—Position for examining lungs posteriorly in older child.

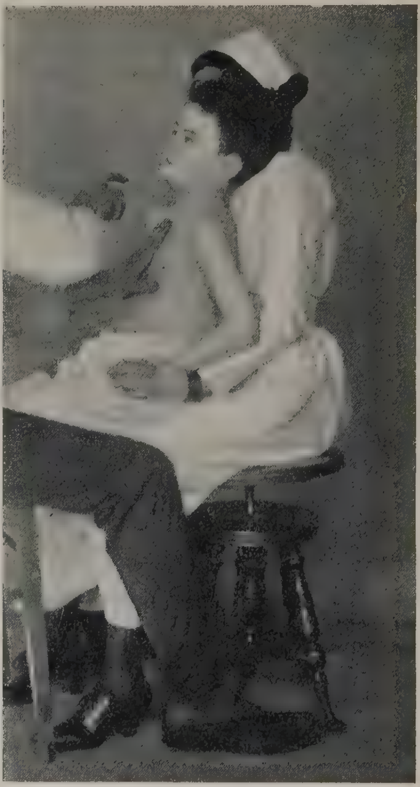


Fig. 30.—Position for examining chest anteriorly.

"Piano" percussion, which consists in tapping the chest directly with the fingertips, is often of value in mapping the heart borders and determining lung resonance. The interpretation depends more on the resistance felt than on the resonance note. Most clinicians can learn more by the resistance felt under the pleximeter finger than by the sound itself, and after experience one can easily determine the various degrees of resistance, whether of resonance, dullness or flatness.

In determining the heart borders in children lighter percussion is employed. The right margin is easily ascertained; the left border of an unusually large heart cannot be definitely mapped out. It should be remembered that normally, in a child, the right border may extend to the right sternal line, and the left one-half of an inch outside the nipple line.

Fluid effusions occur more commonly in the pneumonia of children than in that of adults. A thickened pleura or a thin layer of fluid over a pneumonic area gives a flatter percussion note than a simple pneumonia and this difference may be detected if light percussion is employed. In large effusions and massive empyema no difficulty is found in determining the flatness of the percussion note as would be expected, even on deep, heavy percussion.

Auscultation.—In most cases auscultation gives the best results when the child is quiet, although at times crying during auscultation aids in making the diagnosis, especially in pneumonia and pleural effusions. For obvious reasons the stethoscope should always be warmed before it is applied. Most children not only become accustomed to the stethoscope but anticipate with pleasure the return of the doctor with his "telephone."

As in adults we note the character of the breath sounds, the normal respiratory murmur, the vocal resonance, and the presence of pathologic breathing. Unless one is experienced in the examination of infants and young children great difficulty is encountered in the auscultation of the lungs. The thinness of the chest wall, the shallowness and rapidity of the respiration and the comparatively large size of the bronchi combine to make the respiratory murmur louder, sharper and harsher than in the adult.

Bronchial breathing in children is similar to that of adults. We commonly hear the term "roughened breathing" or the statement that the breathing is almost bronchial. These expressions are misleading and may cause the physician to fall into error. Bronchial breathing is in no way to be compared with this type of breathing and the term "roughened breathing" or "increased breathing" should be abolished. A much better expression is "roughened respiratory murmur." Bronchial breathing if present is unmistakable; whenever we are in doubt the breathing is probably not bronchial. Bronchial breathing depends in no way upon loudness or roughness. In fact if heard through an effusion it may be scarcely audible. Under normal conditions the expiratory sound is barely perceptible. In bronchial breathing the expiratory breath sound is markedly prolonged and of the same character as the inspiratory sound. Teachers frequently observe that classes of students beginning the examination of children suffering from pneumonia diagnose pneumonia over the healthy areas and *vice versa*. What they suppose to be bronchial breathing is the intensified, loud, rough, respiratory, compensatory murmur of the healthy lung which is called upon to do the work of both lungs.

Bronchial breathing in children does not always signify lung consolidation, for we not infrequently observe it in a massive empyema.

In many cases of pneumonia in young children the breath sounds instead of showing the customary bronchial character are distant or absent. Distant bronchial breathing is usually due to pleurisy superimposed upon pneumonic consolidation.

Auscultation of the heart is practiced in children as in adults. We note the character of the heart sound, its rate, rhythm, tone, loudness and relative accentuation or diminution in the different areas. Fright or other emotional disturbance cause temporary accentuation of the second pulmonic sound and for this reason accentuation of this sound is of less diagnostic importance than in adults. A duplication of the second sound may be heard in children under normal conditions. The presence of murmurs and friction rubs should be determined. Their location and the point of greatest intensity, the time, whether systolic or presystolic or diastolic, their character, whether loud, soft, blowing, harsh or musical, the direction of their transmission, whether toward the axilla, or toward the great vessels of the neck, must be carefully noted. Murmurs may be functional or organic due to congenital malformations or acquired valvular disease. Functional murmurs occur less commonly in children than in adults. Functional murmurs are always systolic and are heard most frequently to the left of the sternum although, occasionally they may be heard at the apex. They are always soft, blowing in character and are usually observed in anemic, emaciated children or those suffering from some febrile disease. The heart is never enlarged in this condition and the other tones are usually unaffected. Functional murmurs rarely occur under four years of age and the same may be said of the organic valvular lesions. In early childhood congenital heart disease is the most common condition encountered.

Examination of the Mouth and Throat.—A complete examination of the mouth and throat should be made in every case brought to the physician, regardless of the complaint, whether the symptoms point in that direction or not. Many obscure diseases of unknown origin may be cleared in this way. It is a well known fact that children with most severe mouth and throat infections may not indicate the site of their trouble in any manner except possibly by the refusal of food. Children suffering from tonsillitis of the severest type, or even diphtheria, may be treated for days for other troubles, chiefly gastrointestinal unless a local examination is made. We should never attempt to make an examination of the mouth and throat until the child is held properly and the light properly adjusted. The mother should face the window or the light with the child on her lap facing in the same direction. One hand should encircle the child's waist and hold its hands fast, the other should hold the child's head against the mother's shoulder. She should be cautioned to hold the child tightly, for the tendency of most mothers is to release her hold when the child struggles and the struggle is usually unavoidable.

It is often convenient to bind the arms of the child to his side with a blanket or towel, as this makes the mother's work easier. The examination should be done quickly and in most cases should require only a second or two. An old fashioned spoon handle or a tongue depressor of similar shape is most advisable and works much more efficiently than the ordinary flat spatula.

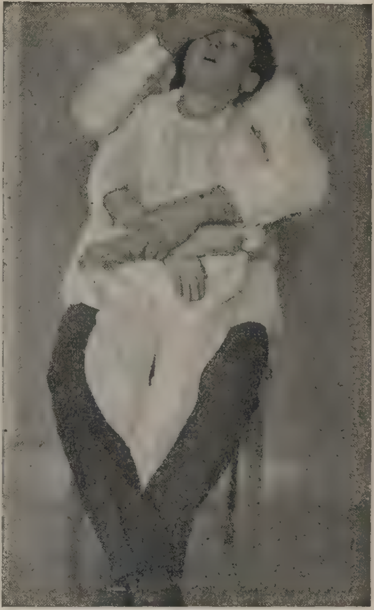


FIG. 31.—Method of holding child for nose, mouth and throat examination.

Examination of the Nose.—Nasal obstruction and discharge are the most frequent pathologic nasal conditions observed. Acute obstruction is usually due to a bacterial infection. This discharge may be caused by a variety of microörganisms, chiefly the pneumococcus and streptococcus. A profuse nasal discharge causing excoriation of the skin, especially if blood tinged, should always suggest diphtheria or syphilis, as we have said before. A profuse, purulent, blood-tinged discharge coming from one nostril usually means that a foreign body is imbedded within. Nasal obstructions, even mild coryzas, cause much discomfort. Headache, dyspnea, restlessness and even loss of appetite result. Chronic obstruction in very young infants usually suggests syphilis. Snuffles is at times so characteristic as to make the diagnosis positive. In older children adenoids are usually the seat of the trouble and are best determined by digital palpation of the nasopharynx.

The Nasopharynx.—The nasopharynx of young children and infants cannot be examined successfully with a mirror. We must depend almost entirely upon the character of the voice, upon the type of breathing, and digital examination. In acute nasopharyngitis it is not advisable to make a digital examination, but in chronic cases when adenoids are suspected this procedure must be followed if the condition is to be learned definitely. The child should be held, as previously described, as in all mouth and throat examinations. The physician stands at the child's right side and with his left hand holds the mouth open with his finger, if no teeth are present, or with a mouth-gag if the child has teeth. The right forefinger is passed gently into the mouth pointing upward and hooked under the soft palate, then gently swept around.

Examination of the Ear.—The examination of these organs in children is of greater importance than is usually recognized. Whenever a child suffers from a fever which cannot be explained by any local condition the ears should be examined. It is well known that otitis is one of the commonest causes of fever of apparently obscure origin.

In all acute infectious diseases the ears should be frequently examined. We know that otitis media is a common complication in scarlet fever, pneumonia and in all respiratory diseases. In some epidemics of grippe almost every child suffers from otitis media as a complication. Whenever the temperature lasts longer than is usual the cause may frequently be discovered by an examination of the drum membrane. We should not forget that young children frequently manifest no symptoms of local trouble In some cases there may be fever and severe pain followed by an unexpected discharge from the ear. In some cases fever even may be absent and in others the child may not cry from pain, the first symptom being the discharge of pus or blood. On the other hand our attention may occasionally be drawn to the ear of the infant. The peculiar cry, the tugging or pulling at the ears or the placing of the hand to the head may give us a clue. Various inflammations of the drum may be noticed, varying from a slight reddening of the membrane to a marked bulging. In infancy the drum is more horizontal than in older children.

In examining the ears of children an especially small speculum should be used. Many find it more satisfactory to examine the child's ear without a speculum, the canal being straightened by pulling the auricle forward and upward and the ear downward and backward. In older children the ear should be pulled upward and backward as in adults.

Retropharyngeal Abscess.—A child suffering from retropharyngeal abscess cries in a peculiar manner, as though a hot potato were held in the mouth. He holds his head backward rigidly or to one side and shows evidence of obstructed respiration and difficulty in swallowing. As a rule the glands surrounding the neck are enlarged. In every case showing symptoms of retropharyngeal abscess, digital examination should be made, for in this way only can a diagnosis be arrived at. The finger should be passed gently over the base of the tongue to the posterior wall of the pharynx. The swelling is evidenced by a more or less fluctuating mass on either side of the median line on the posterior wall of the pharynx. We should not forget that there are two retropharyngeal glands, and the swelling is usually unilateral.

Rectal Examination.—A rectal examination is indicated in all abdominal conditions in which the diagnosis is not clear. In suspected appendicitis, tuberculous peritonitis, or intestinal obstruction, digital examination should always be part of the general routine. Tenderness in the abdomen and pelvis, abnormalities and tumors, may be disclosed by a rectal examination.

The child should lie on its back with the thighs flexed on the abdomen. The physician stands to the right of the patient and slowly introduces his right forefinger, which should be well lubricated, into the rectum. The left hand palpates the abdomen while the finger sweeps round and explores the rectum.

The Temperature.—In a child under six years of age the temperature should always be taken by rectum. In older children it may be taken by mouth. Temperatures in the axilla and groin are unreliable and present no advantages. As a rule the child is frightened by the thermometer and it is best to take the temperature near the end of the examination. The normal temperature of a child should be between 98.6 and 99 degrees. It is commonly supposed that the temperature by rectum is one degree higher than in the mouth. The difference is probably not more than 1/5 to 3/5 of a degree. Some children under normal conditions persistently run a fever course as high as 99½ degrees and occasionally even higher. The heat regulating centers in the brain are more easily affected in children than in adults and children run high temperatures from apparently slight causes.

GENERAL CONCLUSIONS

The child can no longer be considered as an adult on a small scale. He is anatomically and physiologically a distinct entity. Disease attacks him in a different manner. The prognosis differs. The clinical course follows separate paths. These factors vary also during the various ages of childhood. Pediatrics is a special study. Each individual child, sick or well, is also a special study.

To cover the entire field of physical diagnosis, history taking, and examination in a single chapter is impossible. It has been attempted merely to formulate a method of approaching the problem in a more or less systematic manner. Practice and experience will establish rules, but the clinician's individuality will govern their application in the examination of infants and children.

CHAPTER XIV

CEREBROSPINAL FLUID

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The study of cerebrospinal fluid, although important in all branches of medicine, is doubly important in pediatrics; for one who is concerned with diseases of children uses the cerebrospinal fluid more often for diagnostic and therapeutic purposes than one engaged in any other line of medicine. In this chapter, particular emphasis will be laid on cerebrospinal fluid in its relation to pediatrics.

History.—It is highly probable that the ancients, who held the theory that some fluid (humor) dripped down from the brain into the throat knew of the existence of cerebrospinal fluid. However, one does not find an accurate description of the fluid in any of the ancient writings. Some credit the first authentic description of cerebrospinal fluid to Contugno (1764); others claim it for Valsalva; Haller, in his book, *Physiology of Man*, written in 1766, makes mention of its presence, and Magendie (1825) gives a full and accurate description of the fluid. Since Magendie's time numerous workers have enriched our knowledge on the subject, and have shown its importance in physiology and pathology.

NORMAL CEREBROSPINAL FLUID

Although no one has ever determined the exact amount of cerebrospinal fluid in the body during life, a procedure that would hardly be possible, the amount of fluid present in the human body is estimated to vary from 25 to 372 c.c. Post-mortem, Magendie found 62 c.c. as the average amount of cerebrospinal fluid in a medium sized man.

The cerebrospinal fluid is contained in the ventricles of the brain, and in the subarachnoid space of the brain and cord. Magendie found 20 to 30 c.c. of fluid in the ventricles and 32 to 42 c.c. in the subarachnoid space of the brain and cord. The controversy as to whether the fluid in the ventricles communicates with that in the subarachnoid cavity may be considered as settled in the affirmative. The communication takes place through the foramen of Magendie at the posterior part of the fourth ventricle and through the foramina of Luschka on the sides of the fourth ventricle.

The origin of the cerebrospinal fluid is a matter that has not been definitely settled. The prevailing view in the literature, however, is that the fluid originates from the chorioid plexus, a convoluted system of blood vessels in connection with a fold of pia mater which is prolonged into the ventricles and which is covered by an epithelial layer.

The absorption of the fluid is another question not yet definitely established. There is evidence pointing toward a double route of absorption: (1) into the blood stream proper all along the brain; (2) into the perivascular spaces, the lymphatic system draining into the cervical glands, perilymph space of the labyrinth, and lymph vessels of the nose.

The function of cerebrospinal fluid is to protect the nervous system from trauma by equalizing the pressure in the cerebrospinal axis. Among the other functions attributed to cerebrospinal fluid are the following:

1. That it is a primitive digestive juice (Gaskell).
2. That the cerebrospinal fluid is concerned in respiration, and that the chorioid plexus fulfills the functions of an intracerebral gill (Dandy).
3. That the fluid has an internal secretion (Petit and Girard).
4. That in circulating in the perivascular and pericellular channels, the fluid gives up water and carbon dioxide and takes up oxygen and sugar (Mott).
5. That it is a Locke modification of a Ringer's solution which bathes the neurons (Halliburton).
6. That the cerebrospinal fluid contains ferments which protect the nervous tissue against infection (Kafka).
7. That it is a dialysate of the plasma on specially differentiated epithelium, analogous with the aqueous humour of the eye (Mestrezat).

Normal cerebrospinal fluid is clear and colorless. It has a low specific gravity, a low viscosity, and contains very few cells. Qualitatively, its chemical composition is the same as that of blood, with minor exceptions. Quantitatively, it differs from blood in that many chemical ingredients are present in smaller proportions. The water content of cerebrospinal fluid varies between 98.602 to 99.124 parts per 100. The total solids vary between 0.876 to 1.398 parts per 100. The protein varies between 0.013 to 0.047 gm. per 100 c.c. The sugar varies between 0.06 to 0.1 per cent. The chlorides vary between 0.040 to 0.75 per cent. The chlorides exist mostly in combination with sodium, the relation between potassium and sodium being 1:18.

The reaction of cerebrospinal fluid is the same as that of blood, being just above the neutral point toward the alkaline side, running between a P_H of 7.4 to 7.6, if the fluid is examined immediately after removal from the body. The alkali reserve of the fluid is also the same as that of the blood, varying between 45 and 63 per cent. CO_2 at 0° temperature, at 760 barometric pressure.

PATHOLOGICAL CEREBROSPINAL FLUID

From a pathological standpoint cerebrospinal fluid may be divided into three classes: (1) Fluid in metabolic conditions (2) fluid in chronic conditions affecting the nervous system in general, and (3) fluid in acute meningeal conditions. The changes in metabolic conditions are usually only chemical in nature, such as an increase in urea in uremic coma, and an increase in sugar in diabetes. The changes in conditions affecting the nervous system are much more extensive, being physical, chemical, cyotological and immunological.

Acute meningeal conditions are produced either by an irritation of the meninges due to pressure from the brain cavity (tumor, abscess), irritation by circulating toxins (meningism) or by an inflammation of the meninges proper (meningitis). The latter is usually produced by bacteria invading the meninges.

In irritation of the meninges the changes in the cerebrospinal fluid are usually physical and occasionally cytological in nature. In all forms of meningitis the changes are more pronounced.

In the following pages, we shall describe the various tests used for the detection of pathological changes, and discuss their significance.

Physical Changes.—The most important physical changes taking place in pathological cerebrospinal fluid, are an increase in amount and pressure, change in color, formation of foam on shaking, formation of a sediment on standing.

Amount.—Even in adults, cerebrospinal fluid is increased in amount in all conditions affecting the nervous system. In children, this phenomenon is much more pronounced. The child's nervous system is so unstable that many diseases, infectious or otherwise, irritate the nervous system and affect the mechanics of the cranial cavity, and this in turn influences the amount of cerebrospinal fluid in the ventricles and subarachnoid space.

Clinically, this irritation often manifests itself in the form of convulsions, frequently observed at the onset of many infectious diseases, and in disturbances of the gastrointestinal tract.

No special apparatus is required to measure the amount of the fluid removed by spinal puncture; anything above 10 c.c. removed at one sitting indicates an increase in amount. In most infectious diseases, especially those affecting the nervous system primarily, the amount of cerebrospinal fluid one is able to remove by spinal or ventricular puncture often runs as high as 30 to 50 c.c.

Pressure.—With an increase in the amount of cerebrospinal fluid, there is a corresponding increase in the pressure of the fluid. Roughly, an increase in pressure can be noted by the naked eye by watching the force with which the cerebrospinal fluid comes out from the subdural canal. Anything over 10 drops per minute is considered abnormal. For more accurate measurement a special apparatus is needed. Several apparatus have been described for that purpose. Quincke has used an apparatus consisting of a bent glass tube 1 mm. in bore attached to the puncture needle, the pressure being expressed in millimeters of water. Skoog described a mercury manometer for that purpose. We have used a pressure apparatus which allows no loss of fluid before the manometer is attached. One thing must be remembered, and that is that the measurement of pressure expresses only the force with which the fluid comes out from the spinal canal, but does not give any actual measurement of the intracranial pressure, although it is obvious that whenever the pressure measuring the force of withdrawal of fluid is increased, the fluid has also been under great pressure in the cranial cavity and in the subarachnoid space.

The normal pressure in children is given by Quincke as varying between 40 to 60 mm. of water in height. We found it to vary between 45 to 90 mm. of water when the child is in the recumbent posture and between 150 to 230 mm. of water when the child is in the sitting posture; crying, coughing and struggling increase the pressure. The exact mechanism regulating the cerebrospinal fluid pressure is not understood. One factor, however, is known to exert a great influence, and that is the blood pressure in the cranial cavity. Anything that raises the blood pressure in the cranial cavity raises the cerebrospinal fluid pressure, and *vice versa*.

Color.—Many pathological conditions of the meninges, brain or cord produce a change of color in the cerebrospinal fluid. In all suppurative meningitides the fluid is turbid, varying in shade from mere opalescence to a pearly gray or greenish-yellow color. In cases of tuberculous meningitis, the cerebrospinal fluid is usually clear, although at times it becomes slightly opalescent. The same is true of poliomyelitis. In jaundice, the cerebrospinal fluid is usually greenish yellow in color. In the condition designated as xanthochromia, the fluid is deep yellow in color, which together with the characteristic massive coagulation and the large amount of protein indicates compression of the spinal cord (Froin's syndrome).

Although no special apparatus is needed for the observation of color, it is advisable to note a few points: The tubes used for the collection of the fluid should be made of thin glass and of equal size, and should always be kept in the same position to the light during the examination. Daylight is naturally better for the observation of color than is artificial light.

Foam.—We noticed the formation of a foam in all pathological fluids. When a test tube is half filled with normal cerebrospinal fluid and shaken for two or three minutes, a thin foamy layer forms, which disappears in a few minutes. In all pathological cases, however, a thick foam, one-half to two inches in height, appears and persists from 30 minutes to several hours.

This foam formation, although not conclusive, serves as an indication of a pathological cerebrospinal fluid.

Sediment.—In most if not all cases of acute meningitis, a pellicle or sediment, consisting mostly of fibrin and leucocytes, is formed. In suppurative meningitis, the pellicle forms in a very short time. In tuberculous meningitis it forms in a period varying from 12 to 24 hours. The only condition required for the formation of a pellicle is that the test tube containing the fluid be left undisturbed.

The determination of freezing point and surface tension have been used by some to differentiate normal from pathological cerebrospinal fluid, but as yet the value of these factors in diagnosis has not been definitely established.

Chemical Changes.—The chemistry of cerebrospinal fluid has, of late, been made the subject of thorough investigation, and many facts of diagnostic importance have been brought out in the study. The most important chemical tests utilized for diagnosis are the following: globulin, organic index, sugar, and chlorides.

Globulin Tests.—All inflammatory processes of the meninges produce a high globulin content in the fluid. This increase of globulin can usually be detected by qualitative tests. The more acute the inflammation, the greater the increase in the globulin. With the exception of the sulphosalicylic mercuric chlorid test which gives a characteristic precipitation, the globulin tests are of value only in detecting the presence of an inflammatory condition of the meninges, but they give no indication as to the specific type of the lesion. Many globulin tests have been proposed, but for clinical purposes the Pandy, Noguchi, Ross-Jones, and Nonne are most commonly used.

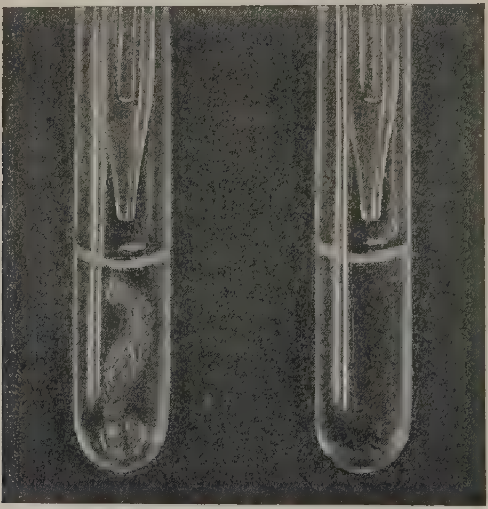

FIG. 32.—Pandy test. A, positive; B, negative reaction.

Pandy.—One drop of cerebrospinal fluid is added to 1 c.c. of a concentrated solution of carbolic acid (1 part of phenol crystals to 15 parts of water). A bluish-white ring or cloud results if globulin is present in excess of normal (Fig. 32).

Noguchi.—Two-tenths cubic centimeter of cerebrospinal fluid is poured into a small test tube, preferably a Noguchi tube, and 0.5 c.c. of a butyric acid solution (5 c.c. of butyric acid to 45 c.c. physiological salt solution) is added to the fluid. The mixture is boiled for a few seconds and 0.1 c.c. of NaOH (normal 4 per cent. aqueous solution) is added after which it is boiled again for a few seconds. If the globulin in the fluid is increased, a fine or coarsely granular, flocculent deposit forms in from 10 to 20 minutes. If no coarse flocculi appear within two hours, even if a slight opalescence is present, the glubolin is not increased.

Ross-Jones.—Two-tenths to 0.5 c.c. of a saturated ammonium sulphate solution is transferred to a Noguchi tube. An equal amount of cerebrospinal fluid is floated upon it by running it down the side of a slanted tube. A white ring develops in a few minutes at the point of contact, if the globulin in the fluid is increased.

Nonne-Appelt.—Phase I.

Same as Ross-Jones except that equal parts of cerebrospinal fluid and saturated ammonium sulphate are mixed instead of being superimposed one upon the other. A white precipitation forms in three minutes if the globulin is increased.

Phase II.—The precipitate is filtered and one drop of dilute acetic acid (10 per cent.) is added to the filtrate and boiled. A precipitation forms if the fluid is pathological (serum albumin).

Characteristic Precipitation.—Tashiro and the author found that if an alkaloidal and metallic precipitant are added to two different portions of the same specimen of cerebrospinal fluid, a characteristic ratio will be obtained in certain pathological conditions. We used a 3 per cent. solution of sulphosalicylic acid as the alkaloidal precipitant and a 1 per cent. solution of mercuric chlorid as the metallic precipitant. One cubic centimeter of cerebrospinal fluid was added to both solutions.

Normal fluid was found to become slightly turbid with sulphosalicylic and remain clear with mercuric chlorid. Standing with either reagent produced very little sediment at the bottom of the tube, the sulphosalicylic precipitate measuring 1 to 3 mm. and the $HgCl_2$ measuring 3 to 4 mm. in height in a tube 7 to 8 mm. wide.

In tuberculous meningitis, the sulphosalicylic acid gave upon immediate examination a turbid precipitate which settled to the bottom and in several hours reached a height of 3 to 6 mm. The mercuric chlorid was only slightly turbid at first, but in 12 to 24 hours it formed into a gelatinous precipitate measuring 6 to 20 mm. in height.

In meningococcus meningitis, before the administration of serum, the sulphosalicylic acid produced a very turbid color, immediately forming a heavy precipitate, which in several hours settled to a height of from 7 to 20 mm. The tube with mercuric chlorid, on the other hand, was clear immediately, and showed but a very slight sediment on standing, ranging from 1 to 7 mm. in height. The ratio between sulphosalicylic and mercuric chlorid on standing, therefore, serves as a means of diagnosis, not only indicating the presence of a pathological condition, but showing the character of the meningitis, whether, for instance, it is meningococcic or tuberculous in type (Fig. 33).

The quantitative determination of the protein in the cerebrospinal fluid can be made in more than one way. It may be done by determining the total and non-protein nitrogen content of the fluid, and finding the protein figure from it, or various modified albuminometers may be used. One of the simplest modes of procedure is to use a modified Esbach tube, with smaller amounts of cerebrospinal fluid and of the Esbach reagent than is used for urinalysis, the resultant precipitate being read at the end of 24 hours.

Organic Index.—This test is used for the purpose of determining whether or not the organic substances in the cerebrospinal fluid are increased above normal. It was originally used by Kubel-Thiemich for the determination of organic substances in water, and later applied to cerebrospinal fluid by Mayerhofer, who showed that cerebrospinal fluid oxidizes, or, as he termed it, reduces permanganate and that fluid from cases of meningitis has a higher oxidation index than normal fluids or even those of meningism. The technique of the test is as follows:

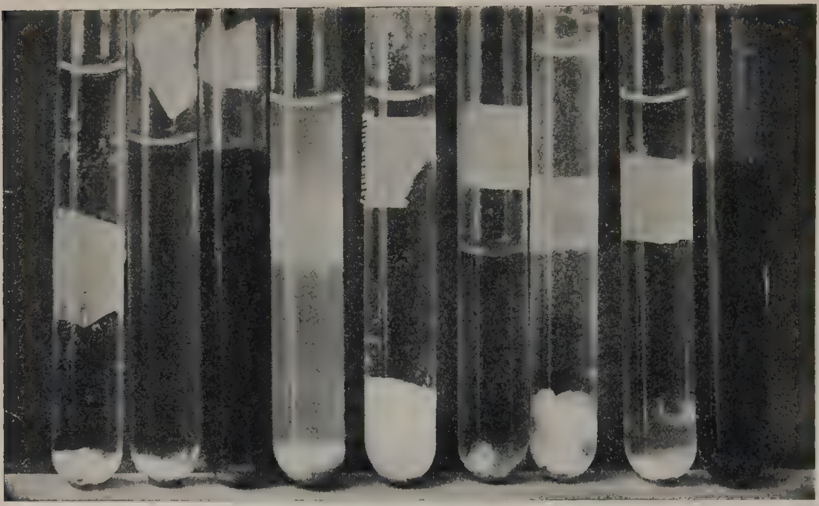

Fig. 33.—Photograph showing the typical ratio of precipitation of the two different precipitating agents.

1. 1 c.c. of non-meningitic fluid plus 1 c.c. of 1 per cent. $HgCl_2$.
2. 1 c.c. of non-meningitic fluid plus 1 c.c. of 3 per cent. sulphosalicylic acid.
3. 1 c.c. of non-meningitic fluid plus 1 c.c. of phenolphthalein.
4. 1 c.c. of fluid from meningococcus meningitis plus 1 c.c. of $HgCl_2$.
5. 1 c.c. of fluid from meningococcus meningitis plus 1 c.c. of sulphosalicylic acid.
6. 1 c.c. of fluid from meningococcus meningitis plus 1 c.c. of phenolphthalein.
7. 1 c.c. of fluid from tuberculous meningitis plus 1 c.c. of $HgCl_2$.
8. 1 c.c. of fluid from tuberculous meningitis plus 1 c.c. of sulphosalicylic acid.
9. 1 c.c. of fluid from tuberculous meningitis plus 1 c.c. of phenolphthalein.

One cubic centimeter of the cerebrospinal fluid is introduced by means of an accurately graduated pipette into an Erlenmeyer flask; 50 c.c. of distilled water and 10 c.c. of diluted H_2SO_4 (one part of H_2SO_4 and three parts of H_2O) are added and the mixture is brought to the boiling point; 10 c.c. of a decinormal permanganate solution is then introduced into the flask and the solution is boiled for exactly 10 minutes. At the end of this time, 10 c.c. of decinormal oxalic acid is put into the flask, whereupon the red or yellowish-red color turns white. Titration is carried on drop by drop from a burette containing decinormal permanganate solution until the color of the solution in the receptacle turns red and remains so for a number of minutes. The number of cubic centimeters of permanganate required to produce the end reaction is then read off, and the figure is taken as the permanganate index. A permanganate index of two (after subtracting the amount needed for the reduction of water) is taken as the standard. An index higher than two, especially if the index in the first portion of the fluid is higher than in subsequent ones, is indicative of meningitis. An index lower than two, particularly if the subsequent portions are higher than the first one, speaks for meningism.

A number of investigators have employed the permanganate test; some consider it of great value, and others of very little. Although it occasionally fails, it often furnishes a clue to the high content of organic matter in the fluid, which is of value toward establishing a diagnosis.

Sugar.—The reducing substance in normal cerebrospinal fluid was first observed by Claude Bernard, who considered it a dextrose. The nature of the reducing substance had, however, been disputed by several men, notably Halliburton. Now, however, there is no longer any doubt of its being a dextrose. The amount of sugar contained in normal cerebrospinal fluid has been found to vary from 0.06 to 0.1 per cent. This amount is increased in diabetes mellitus, decreased in some forms of meningitis, and usually absent in the severe forms of meningococcus meningitis.

The qualitative determination of sugar in the fluid by Fehling solution is not delicate enough, since by this method only an absence of sugar, but not a decrease in its amount, can be detected. The quantitative determination should, therefore, be used whenever possible.

Several methods have been employed for sugar determination, the most accurate one being that of Lewis and Benedict, which was originally described for blood sugar. The technique is as follows: 2 c.c. of cerebrospinal fluid is put into a 25 c.c. volumetric flask containing 5 c.c. of distilled water. 15 c.c. of saturated picric acid solution is added, water filled up to 25 c.c. and the whole solution is shaken and filtered. Eight cubic centimeters of the filtrate in duplicate is put into large Jena test tubes for determination. Two cubic centimeters of saturated picric acid solution and 1 c.c. of a 10 per cent. sodium carbonate solution are added. The content of the tube is evaporated over the flame until precipitation occurs. Three cubic centimeters of water is added, and the tube heated again to the boiling point, in order to dissolve the precipitate. The content of the tube is now transferred to a 10 c.c. volumetric flask, cooled, filled up to the mark with water, shaken, and filtered through cotton into a colorimetric flask. The color is compared in a Duboscq colorimeter either with a dextrose standard, freshly made up every time, or with a permanent standard consisting of 0.064 mg. picramic acid and 0.1 gm. sodium carbonate in 1000 c.c. and the results are calculated.

Epstein modified the Lewis Benedict method, using a solution of picramic acid in a sealed tube as a standard. Although not as accurate as the original method, it serves the purpose. Other quantitative blood sugar methods may also be used for the determination of sugar in cerebrospinal fluid.

Chlorids.—Normal cerebrospinal fluid contains 0.6 to 0.74 gm. of chlorid (NaCl) per 100 c.c. In edema of the brain, especially in tuberculous meningitis the amount of chlorids in the fluid is diminished. The determination of chlorids thus becomes one of the diagnostic tests for certain types of meningeal involvement. The method that lends itself best for the determination of the chlorids in the cerebrospinal fluid is that of Seelman, originally described for the determination of chlorids in urine. Two standard solutions, Solution 1 and Solution 2, are used. Solution 1 consists of 29.055 gm. of anhydrous crystallized silver nitrate (C.R.); 900 c.c. of a 25 per cent. nitric acid (in distilled water); 50 c.c. cold saturated solution of ammonio-ferric alum in distilled water, and a sufficient quantity of water to make up to 1,000 c.c. Solution 2 consists of 7 gm. of ammonium sulphocyanate dissolved in 1,000 c.c. of distilled water. The solutions must be so standardized that the last drop of Solution 2 when added to 1 c.c. of Solution 1 will bring about the end reaction, which is manifested by a reddish-brown color.

The determination of chlorids in the cerebrospinal fluid is made as follows: 0.5 c.c. of cerebrospinal fluid to be tested is placed in a porcelain dish, 1 c.c. of Solution 1 is added and the mixture is stirred for about a minute with a glass rod. Solution 2 is now added drop by drop by means of a 2 c.c. pipet graduated to at least 0.05 of a c.c. and the mixture is stirred until the brown color developing after each drop disappears. The amount of Solution 2 which has been used to bring about the end reaction is now read off, and the difference between the amount used and 2 c.c. is equal to the number of grams of sodium chlorid per 100 c.c. of the specimen tested. If, for example, it takes 1.26 c.c. of Solution 2 to bring about the end reaction, the amount of chlorid in 100 c.c. of cerebrospinal fluid equals $2-1.26$ which equals 0.74 per cent. of chlorids.

Lactic acid is present in traces in normal cerebrospinal fluid, and is increased in all suppurative meningitides, and according to one author, also in every case showing edema of the brain. Lactic acid, however, cannot be used for diagnostic purposes. The same can be said of acetone, which is absent in normal fluid and present in the fluid of diabetics and of those suffering from suppurative meningitis.

Physicochemical.—The recent advance of physical chemistry has left its impress on the study of cerebrospinal fluid, furnishing us with a number of tests based on physicochemical principles. Among these may be mentioned the Lange colloidal gold reaction, the mastic reaction, the cataphoresis reaction and the Ninhydrin reaction. While Lange himself considered the colloidal gold reaction to be simply an indication of increased protein in the cerebrospinal fluid, we believe that it is the physicochemical properties of specific proteins that are responsible for the characteristic curve obtained in certain pathological conditions. The same, we believe, is true in regard to the mastic. Cataphoresis shows the electrical charges of the protein in the cerebrospinal fluid of various meningitides. The Ninhydrin reaction is based on the same principles as the Abderhalden reaction.

The Colloidal Gold Reaction.—Lange found that normal cerebrospinal fluid when diluted with a 0.4 per cent. solution of sodium chlorid does not affect colloidal gold, while pathological fluids in sodium chlorid solutions do change the color of colloidal gold, producing characteristic changes for various diseases. The gold chlorid reaction is now considered specific for the various forms of syphilis of the nervous system and diagnostic of the presence of meningitis. There is one characteristic of the Lange reaction that makes the test different from practically all other tests on cerebrospinal fluid, namely, that the Lange test does not have to be done immediately after the removal of the fluid from the body.

The technique of making up the gold chlorid solution is rather difficult, some solutions producing color changes even with normal fluids, and others producing no changes even with distinctly luetic fluids. Care must be taken to see that the solution is transparent, of neutral reaction, and that it is precipitated by 1.7 c.c. of 1 per cent. NaCl solution in one hour. There are several modifications of the original Lange technique of preparing colloidal gold solution, but we believe the following technique to be very satisfactory.

The method is as follows: To 1000 c.c. of doubly distilled water, 10 c.c. of a 1 per cent. gold chlorid solution and 7 c.c. of a 2 per cent. solution of potassium carbonate are added. The mixture is heated to 90°C., stirred vigorously and 5 c.c. of a 1 per cent. formaldehyde solution is added. The solution should at once assume a red color.

The method of testing cerebrospinal fluid with the above solution is as follows: 0.2 c.c. of cerebrospinal fluid is introduced in the first one of a series of ten test tubes containing 1 c.c. of 0.4 per cent. sodium chlorid. Ten dilutions of the cerebrospinal fluid are now made, by taking 1 c.c. of the first tube and transferring to the second, then 1 c.c. of the second and transferring to the third, etc., the dilutions thus ranging from 1 to 10 to 1 to 5,120. An eleventh tube containing 1 c.c. of 0.4 per cent. sodium chlorid without cerebrospinal fluid is used as a control. It is also advisable to run another gold chlorid test on a fluid known to be normal. To each tube 5 c.c. of the colloidal gold reagent (1 per cent. solution of gold chlorid, 2 per cent. solution of K_2CO_3, 1 per cent. solution of formalin) is added. Normal cerebrospinal fluid does not change the original port-wine or cherry-red color of the gold chlorid solution. Pathological fluids, however, change the color of the gold chlorid solution to red blue, violet, blue, pale blue, or colorless. The changes appear in from 5 to 30 minutes and are intensified on standing several hours. The quality of the change produced and the dilution in which it occurs help to determine the type of disease. The change is usually expressed either in curve form or by numerals, the original color of the gold chlorid solution being designated as 0, red blue as 1, violet as 2, blue as 3, pale blue as 4, and colorless as 5. The accompanying charts show the colloidal gold changes in various diseases (3, 4, 5 and 6).

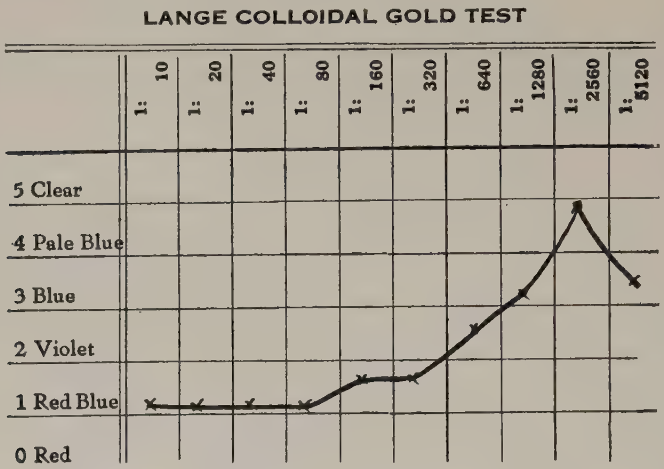

FIG. 34.—Curve in case of meningococcus meningitis.

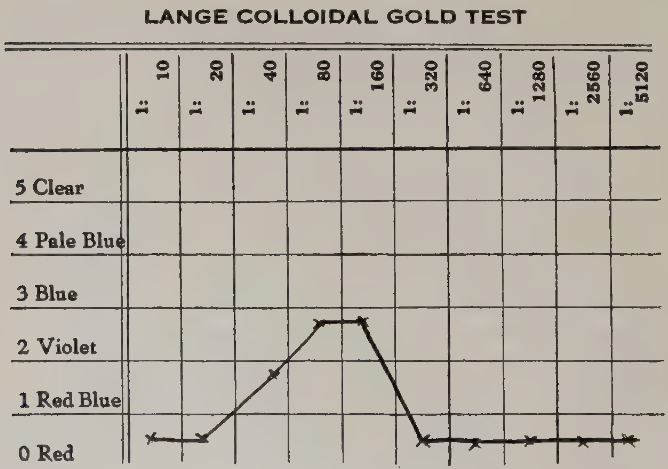

FIG. 35.—Curve in case of tuberculous meningitis.

Mastic Reaction.—The mastic test consists of a reaction obtained by a solution of gum mastic when added to the cerebrospinal fluid.

We believe that the mastic test is not as important as the gold chlorid test, because of the difficulty of interpreting the various shades of opacity encountered. We, therefore, do not consider it necessary to describe the technique here.

Cataphoresis.—Tashiro and Levinson showed the existence of a different charge of the proteins in the fluid of different types of meningitis. We use the apparatus recommended by Michaelis (Fig. 38). The cerebrospinal fluid is placed in tube 3, care being taken to see that there is no bubble inside the core of the stopcocks, tubes 2 and 4 are filled with 3 per cent. sulphosalicylic acid and the upper portions (1 and 5) are filled with distilled water. To A is added $CuCl_2$ and to B is added NaCl. A silver electrode is placed in the anode and a copper electrode in the cathode. The electrodes are connected to a light circuit, 110 volts, with an ordinary lamp in the circuit as resistance. The migration of the protein from the cerebrospinal fluid is detected by the formation of a precipitation in the arm.

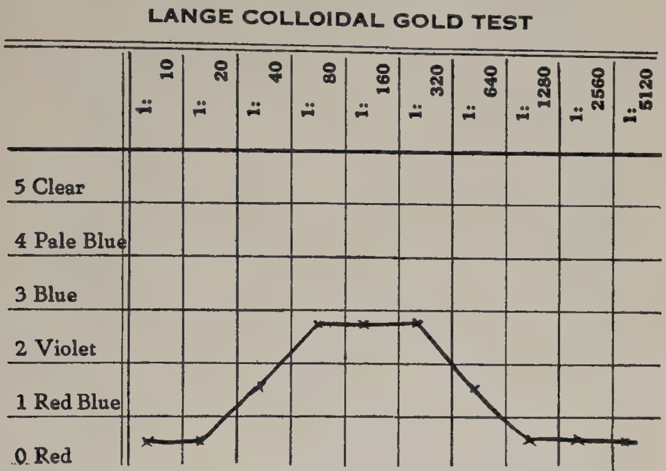

FIG. 36.—Curve in case of tuberculous meningitis.

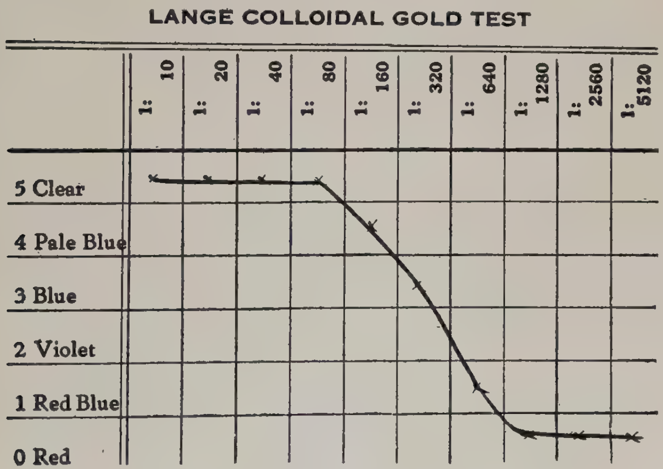

FIG. 37.—Curve in case of juvenile paresis.

We found that in meningococcus meningitis there is a considerable amount of the protein moving toward the cathode, showing the presence of positively charged protein in the cerebrospinal fluid of that disease. In tuberculous meningitis, we found that the precipitate moves toward the anode, showing that the protein in that case is negatively charged. In non-meningitic fluids there is very little precipitation by cataphoresis, only a slight diffusion being seen either at one side or the other.

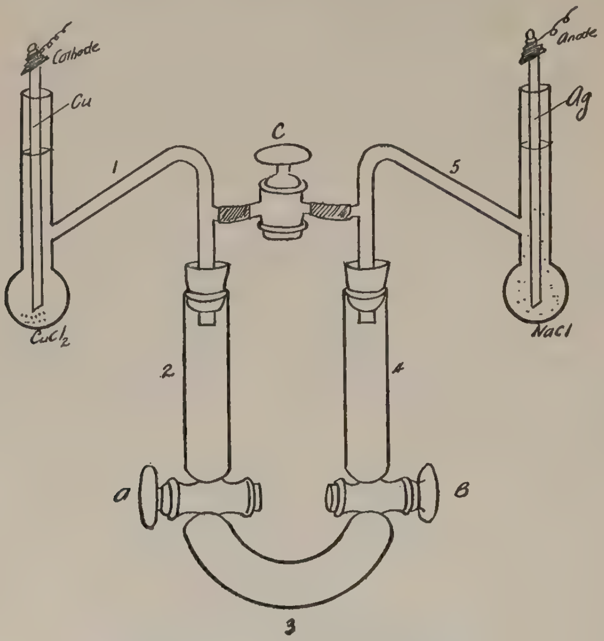

FIG. 38.—Cataphoresis tube.

Ninhydrin Reaction.—Noble found that in tuberculous meningitis a blue to a blue-violet color appears on boiling 0.5 to 1 c.c. of cerebrospinal fluid with 0.1 c.c. of a 1 per cent. ninhydrin solution for about one-half of a minute. Other workers have confirmed his observation.

H-ion Concentration and Alkali Reserve.—Normally the H-ion concentration of cerebrospinal fluid varies between P_H 7.4 to 7.6. In suppurative meningitis we found the H-ion concentration to vary between a P_H of 7.2 to 7.4. Furthermore, the fluid does not become as quickly alkaline as does normal fluid. The same is true of alkali reserve, the alkali reserve in suppurative meningitides being lower than in normal fluid.

Cytological Examinations.—Cytology has proved to be one of the best diagnostic aids in differentiating normal from pathological fluid.

Normal cerebrospinal fluid contains from one to six cells per cubic millimeter, while pathologic fluid contains a much greater number and greater variety of cells. The origin of the cells in cerebrospinal fluid is not well known. Some consider them hematogenic in origin; others consider them histogenic; while still others suggest a dual origin for them, lymphocytes; according to them, being hematogenic and plasma cells histogenic. Whatever the origin of the cells in the cerebrospinal fluid the increase in their number indicates an existing pathological condition in the cerebrospinal canal.

Cytological examination consists of counting the number of cells in the fluid and differentiating the type of cells present.

Cell Count.—A special counting apparatus has been devised by Fuchs and Rosenthal for the counting of cells in cerebrospinal fluid. This chamber is 16 mm. square instead of 9 mm. as in the blood counting chamber, and is 0.2 mm. deep instead of 0.1 mm., as in the blood counting chamber. This chamber, therefore, allows of a smaller error than the blood chamber. In using this chamber the cerebrospinal fluid is drawn up to mark 1 in the leucocyte pipette and the diluting fluid to mark 11. The cells in the whole chamber are counted and the resulting number is multiplied by 11 and divided by 32. As a diluent for the fluid, a solution of methyl violet (methyl violet 0.2 gm., glacial acetic 5 c.c. and water to 100 c.c.) has been found very useful, although ordinary 2 to 3 per cent. acetic acid will do to destroy the red cells in the fluid. The Fuchs Rosenthal chamber, while more accurate, is not absolutely necessary; an ordinary blood counting chamber will suffice. The technique for counting cells in an ordinary leucocyte chamber is as follows: The methyl violet or other diluent is drawn up to mark 1 in the pipette and the cerebrospinal fluid to mark 11. The cells in the whole chamber are counted. In order to obtain the number of cells in 1 cubic mm. of undiluted fluid, the result obtained is multiplied by 11 and divided by 9.

The cells must be counted immediately after withdrawal from the body, as they undergo autolysis on standing. If it is impossible to count cells in the fluid immediately after its withdrawal from the body, the cells may be preserved by the addition of two to three drops of a 3 per cent. acetic acid solution to 5 c.c. of cerebrospinal fluid. The subsequent count will not be very accurate, but will give a good idea of the number of cells in the fluid.

Differential Cell Count.—Under normal conditions, all of the cells in the fluid are small lymphocytes. In pathological conditions the lymphocytes may remain the only type of cell in the fluid, or may constitute only a very small percentage of the cells, the rest being of the polymorphonuclear type. In addition to these two types of cells, endothelial cells are encountered in some pathological fluids. The type of cells may even change at different stages of the same disease. The differential cell count is, therefore, of great significance in the examination of cerebrospinal fluid.

In doing a differential cell count, care must be taken to count only the white cells and not the red or the debris. When the fluid is turbid, as in suppurative meningitis, a differential cell count may be made on an uncentrifuged fluid. If the fluid is clear or only slightly opalescent, it is best to centrifuge the fluid for from 15 to 30 minutes at about 1,500 revolutions per minute. Wright's stain is ordinarily used, although methylene-blue and Gram stain are equally effective. Pappenheim has described a special stain for the cells in cerebrospinal fluid. The stain consists of three parts of saturated aqueous solution of methyl green, and one part of saturated aqueous solution of pyronin. The stain is left on the slide for several minutes. The cells are counted according to their type, and expressed in percentage. Unless the fluid already contains a great deal of albumin, some egg albumin may be rubbed on the slide to prevent the smear from being washed off.

Sometimes it is possible to turn the high power of the microscope on the counting chamber while counting the cells and differentiate between the types of cells present.

Bacteriologic.—The following organisms were found by us to be the most frequent causes of meningitis in children: Tubercle bacilli, meningococci, pneumococci, streptococci, and influenza bacilli.

Table 1.—Frequency of Type of Meningitis in the Michael Reese Children's Hospital, 1908–1920

Tuberculous	125	Streptococcus	3
Meningococcus	89	Influenza	2
Pneumococcus	21	Typhoid	1
Unclassified	32		

The colon bacilli have been reported as the causative organisms of meningitis in the newborn. Besides the above mentioned, syphilitic meningitis is not of infrequent occurrence.

The finding of bacteria, either directly in the cerebrospinal fluid or in culture, is the most positive proof of the nature of the disease. The causative organisms, however, are not always found in the fluid with ease. Meningococcus, for instance, is usually found with ease in direct smear, but does not grow readily in ordinary culture media, requiring ascitic dextrose agar for its growth. The tubercle bacillus is not only detected with great difficulty in the direct smear, but cannot be grown at all on ordinary media. The guinea pig inoculation will determine the tuberculous nature of the fluid. This, however, is usually too late to be of any help in the differential diagnosis of the case. Influenza bacillus is found easily in direct smear, but requires blood agar for its growth. Pneumococcus and streptococcus are usually found very easily, both in direct smear and in culture.

One must be very careful in doing bacteriological work on cerebrospinal fluid, for there are many technical factors that may interfere with the success of the work, such as improper media, insufficient incubation of the organism, contamination by other bacteria, overheating of the slide (protein particles often look like bacteria), and faulty staining methods. It is important to make bacteriological examinations on the cerebrospinal fluid of more than one puncture, even after the diagnosis of the case has been established, as it has been found that at times cases of meningococcus meningitis may show pneumococci in the fluid on subsequent examination.

Direct Smear.—In suppurative meningitis where the cerebrospinal fluid is thick, a slide may be prepared from the uncentrifuged fluid. When the fluid is clear, it should be centrifuged for several minutes, and the sediment examined. Where suppurative organisms are suspected, it is best to stain with both methyl blue and Gram stain. When tubercle bacilli are searched for, the fluid should be allowed to centrifuge at high speed for 30 to 45 minutes and stained by the Ziehl-Nielson method. If a pellicle forms, the pellicle should be stained for tubercle bacilli.

Culture Media.—Several drops to 1 c.c. of cerebrospinal fluid or its sediment are planted on a culture media, the kind of media depending on the bacteria looked for. In cases suspicious of poliomyelitis anaerobic cultures should be made, as it is claimed that in this way the organism will be found in culture.

Immunologic.—Cerebrospinal fluid immunology has proven an invaluable aid both in the diagnosis and treatment of children's diseases. Of the various immunologic methods applied, two are of extreme importance: the agglutination of bacteria, and the complement fixation test known as the Wassermann reaction.

The agglutination test is especially useful in the identification of meningococci and pneumococci in the cerebrospinal fluid, and the Wassermann reaction in the diagnosis of congenital or acquired neurosyphilis in children.

Agglutination of Meningococci.—Two methods have been described for this purpose, the macroscopic and the microscopic.

The macroscopic method is carried out by washing down the growth of the culture in question with 2 to 3 c.c of an 0.8 per cent. of sterile salt solution, and adding 0.2 c.c. of this emulsion to various dilutions (1:10, 1:20, 1:40, etc., to 1:2,000) of antimeningococcus serum, and incubating it for 15 to 20 hours. If the bacteria in the culture is meningococcus, a flocculent precipitate will appear in the tubes, containing as high a dilution as 1:200, or even higher (Fig. 39). If it is not meningococcus, it will either not agglutinate at all, or it will agglutinate only in very low dilutions, such as 1:50, or 1:10.

The microscopic method consists of using one drop of antimeningococcus serum, one drop of whole human blood in a sodium citrate solution (one drop of blood to 2 per cent. sodium citrate in salt solution), and one drop of a suspension of the organism to be tested. This is mixed in a pipette, heated for 20 minutes, spread on a slide, stained and examined under the microscope. If the bacterium is meningococcus, a distinct clumping will be seen under the microscope. This should be controlled by a suspension of meningococcus with normal horse serum, or by a suspension of some other bacteria with antimeningococcus serum.

Wassermann Reaction.—While there are diseases other than syphilis, such as leprosy or sleeping sickness, that give a positive Wassermann reaction, the test is sufficiently reliable to consider the patient syphilitic when the reaction is positive.

The technique of the Wassermann test on cerebrospinal fluid is the same as that on blood, except that the cerebrospinal fluid does not have to be heated as is the case with blood serum; and that the amount of cerebrospinal fluid taken for each test must be greater than the amount of blood used.

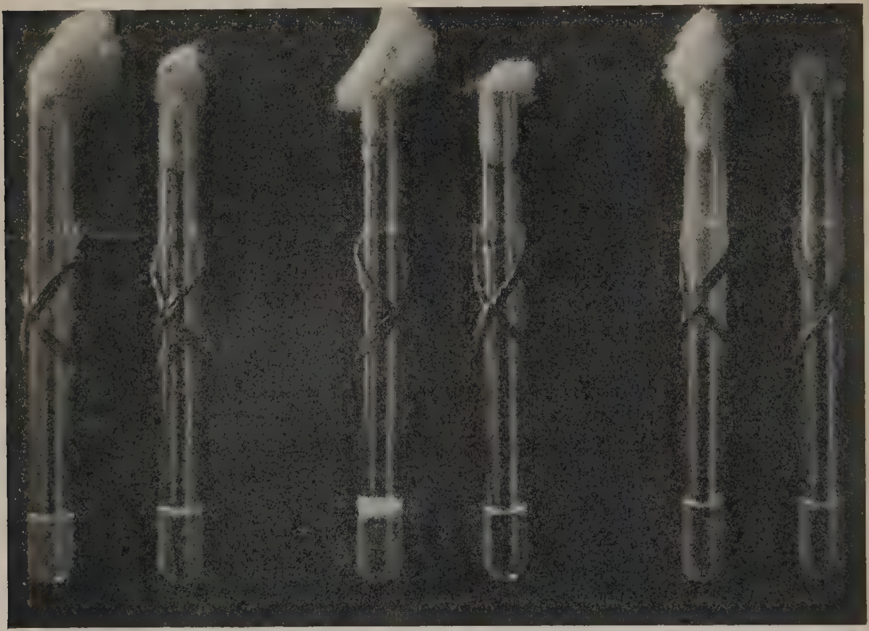

Fig. 39.—Photograph showing agglutination of meningococci by the macroscopical method.

A. Emulsion of meningococci 1:10 dilution of antimeningococcus serum.
B, Control of emulsion of meningococci + salt solution.
C, Emulsion of meningococci +1:160 dilution of antimeningococcus serum.
D, Control.
E, Emulsion of meningococci +1:640 dilution of antimeningococcus serum.
F, Control.

Usually 0.2 c.c. of cerebrospinal fluid is taken for each of the tubes set up, varying the amount of antigen added to the tubes. As a control 0.4 c.c. of cerebrospinal fluid without antigen is used. Schottmüller, however, advises the use of different amounts of cerebrospinal fluid in each of the test tubes. This gives a more or less "quantitative result" as to the degree of positiveness of the fluid.

There is one advantage in doing Wassermann tests on cerebrospinal fluid in children, in that it is possible to obtain large amounts of it, something that is not very easy in doing blood Wassermann in children, unless the longitudinal sinus route be used for the purpose of withdrawing the blood.

Different antigens are employed by different workers. It is best, however, to use three different antigens on the same fluid: cholesterenized, lipoid and luetic. This gives one a triple check on the work.

The complement, the sheep corpuscles and the hemolytic system used for control are the same as those used for blood serum Wassermann.

CEREBROSPINAL FLUID IN VARIOUS DISEASES OF CHILDREN

Cerebral Hemorrhage.—If the hemorrhage is of recent origin the cerebrospinal fluid is bloody. This is not due to the striking of the plexus of veins in the canal, but to the hemorrhage of the brain. At times the fluid contains dried particles of blood, showing that the blood has been in the spinal canal for several days. The cellular content of the fluid consists of both red and white cells with a predominance of red cells. The globulin cannot be determined, due to the dark color of the fluid and to the large amount of globulin naturally present in the blood. If the hemorrhage is of moderately long standing, the cerebrospinal fluid is yellow in color, due to the hemoglobin in it. The cells in such cases are very few in number. Occasionally the cerebrospinal fluid of hemorrhage of the brain is clear, the same as normal fluid. The Wassermann on the cerebrospinal fluid of brain hemorrhage may or may not be positive depending on whether or not the child is syphilitic. The cholesterol of the cerebrospinal fluid is usually increased.

Spina Bifida.—There is usually an increased amount of turbid fluid in the sac and an increased amount of globulin in the fluid. Occasionally, however, the fluid is clear. The cell content of the fluid is not increased.

Tumor of Brain.—Glioma, sarcoma, tubercle, or any other tumor of the brain increases the amount of cerebrospinal fluid so that both the amount and pressure of the fluid removed by lumbar puncture is increased. The fluid, however, may be confined to the ventricles of the brain by a blocking of the foramen of Magendie. In that case the amount and pressure of the cerebrospinal fluid obtained by lumbar puncture is normal. The cells are usually only slightly increased in number ranging between 10 to 12 per cu. mm. The globulin may be slightly increased, although as a rule the globulin tests are negative. The Lange gold chlorid test may show color change in the third, fourth, and fifth tubes. In tumors of the spinal cord the cerebrospinal fluid is usually yellow (xanthochromia) and coagulates spontaneously.

Abscess of the Brain.—What is true of tumor of the brain is also true of abscess of the brain, the fluid being increased in amount and showing no definite signs of inflammation of the meninges. The abscess may, however, break through the brain substance and empty itself in the subdural cavity. In such a case the fluid is turbid and may show the presence of organisms in it.

Hydrocephalus.—The amount of fluid is considerably increased. Most or all of the fluid, however, is contained in the ventricles of the brain. It is not always possible to demonstrate the increased amount of the fluid in the ventricles by lumbar puncture, as the foramen of Magendie often is occluded. The chemical and cytological tests are, as a rule, negative. Occasionally, however, the chemistry and cytology of the fluid are altered. It must be remembered that while hydrocephalus is an entity, it is secondary to some other condition and the fluid in it may undergo changes produced by the primary condition. So does it happen that in hydrocephalus secondary to meningitis, the fluid shows an increased number of cells and positive chemical tests. In hydrocephalus, secondary to lues, the cells are increased, and the Wassermann is positive.

Congenital Lues.—The findings of the cerebrospinal fluid in congenital lues are not constant, depending mainly upon whether or not the cerebrospinal system is involved. When there is an involvement of the nervous system, which is the case in about 33 per cent. of congenital lues, the Wassermann reaction on the cerebrospinal fluid is positive; the cells in the fluid are increased in number and consist mainly of lymphocytes. The protein tests are positive, and the Lange gives a reaction in the syphilitic zone.

In juvenile paresis, the Wassermann is positive in 80 to 100 per cent. of the cases. The cells, which are mainly lymphocytes, are increased, and the protein tests are positive. The Lange curve is characteristic, giving a reaction in the paretic zone.

In syphilitic meningitis, the fluid is increased in amount, so is also the pressure. The leukocytes are increased in number, so is also the globulin. The Wassermann is positive, and the Lange gives a reaction either in the syphilitic or meningitic zone. It is occasionally also possible to demonstrate spirochete pallida by India or Chinese ink.

Anterior Poliomyelitis.—The fluid is increased in amount all through the acute stage and for a week or two during the paralytic stage. The leucocytes during the acute stage are greatly increased, running as high as 300 or 400 per cubic millimeter. During the paralytic stage, the leukocytes are only slightly increased. The type of cell, as a rule, is the lymphoctye, although very early in the disease the neutrophile may predominate. The globulin is increased, as a rule, all through the disease. The Lange shows a fairly constant change in the meningitic zone during the acute stage. Later, however, the changes may occur in the syphilitic zone. An anaerobic micrococcus has been isolated by some authors from the cerebrospinal fluid of anterior poliomyelitis. We have so far been unsuccessful in finding and cultivating the described organism.

Meningism.—In 1894 Dupré described a condition which simulates meningitis, but which later proves to be non-meningitic in nature. This condition is found in pneumonia, otitis media, and other acute infections. Although lumbar puncture has been described by Quincke in 1891, Dupré does not mention this procedure. The diagnosis of meningism, according to him, is to be made by the previous history, by the associated symptoms, and by the urinary phosphates. Now with our easy access to cerebrospinal fluid, our conception of meningism changed. We now understand by meningism cases that show symptoms of meningeal irritation and in which the cerebrospinal fluid is sterile, and shows no increase in the globulin content or only slight chemical changes. In such cases the clinical symptoms and the subsequent history help to differentiate meningism from meningitis.

Tuberculous Meningitis.—In the irritative stage of the disease both the amount and pressure of the cerebrospinal fluid are increased. The amount one is able to remove at one sitting varies between 20 to 50 c.c. The pressure varies between 300 to 700 mm. of water in height. During the paralytic or last stage, when the patient is in deep coma, both the amount and pressure of the fluid are not greatly increased and sometimes even come down to normal limits. The fluid is usually clear all through the disease; occasionally, however, it becomes opalescent. A pellicle forms when the fluid is left standing 12 to 24 hours. This pellicle is usually suspended in the center, and has processes projecting from its sides. It is very delicate and breaks easily. The cells are increased in number, ranging from 15 to 150 per cubic millimeter. Most of them are small lymphocytes, although very early in the disease there may be a preponderance of polymorphonuclear leucocytes in the fluid. The organic substances in the fluid are increased, thus giving a permanganate index higher than two.

The Noguchi, Ross-Jones, and Nonne tests are positive although very early in the disease they may all be negative. Three per cent. sulphosalicylic acid added to 1 c.c. of the cerebrospinal fluid produces a turbid precipitate at first, which settles down later into a small sediment. When 1 c.c. of 1 per cent. mercuric chlorid is added to the fluid, the mixture at first is clear, but later it forms a gelatinous sediment which is greater than that of the sulphosalicylic sediment. The ratio of the two precipitates helps to differentiate tuberculous fluid from that of suppurative meningitis.

The amount of sugar in the fluid equals 0.4 to 0.8 per cent.; only rarely is it altogether absent. The chlorids in the fluid are diminished in amount. The gold chlorid shows a curve in the meningitic zone, the discoloration being mostly in the fifth, sixth, seventh and eighth tubes. Cataphoresis shows the migration of protein toward the anode.

Tubercle bacilli are found in the centrifuged fluid only after careful search (Fig. 40).

Inoculation of the cerebrospinal fluid into a guinea pig usually proves whether or not the case is one of tuberculous meningitis. Five to ten cubic centimeters of cerebrospinal fluid is inoculated into the groin of a guinea pig. A month later, 1 c.c. of tuberculin is injected into the animal. If the meningitis is tuberculous in nature, the guinea pig usually dies the morning after the injection of the tuberculin. Occasionally, however, the animal has to be killed. Tubercles are found in the spleen and inguinal glands on postmortem in positive cases.

Meningococcus Meningitis.—The amount of the cerebrospinal fluid is increased so that, as a rule, from 20 to 50 c.c. can be easily removed by one lumbar puncture. This increase in amount is a constant feature of meningococcus meningitis all through the disease.

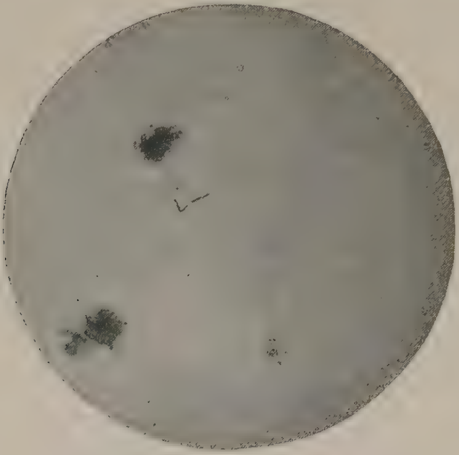

Fig. 40.—Photomicrograph showing tubercle bacilli in cerebrospinal fluid.

The pressure is also increased, ranging between 300 and 700 mm. of of water in height. On standing one-half hour or longer a sediment forms, varying in form and consistency according to the stage of the disease. The cells are greatly increased, running up to as high as 5,000 or 10,000 per cubic millimeter. Ninety-five to ninety-eight per cent. of the cells are polymorphonuclear leukocytes, and many of the cells contain meningococci. Endothelial cells are also usually found in the smear after the second or third day of the disease.

The protein in the fluid is greatly increased, varying between 0.2 to 2 gm. per 100 c.c. All the globulin tests are strongly positive; the ratio of sulphosalicylic to mercuric chlorid precipitate is 2:1, or even greater. Sugar is absent from the fluid or greatly reduced in amount. The chlorids are diminished, but not as much as in tuberculous meningitis, and they may even remain in the normal amount.

The Lange gold chlorid test usually shows a discoloration in the seventh, eighth, and ninth tubes. In the cataphoresis tube, the fluid is usually precipitated with sulphosalicylic acid at the cathode or negative pole. Meningococci are usually found in the direct smear or within the leukocytes. They grow on ascitic dextrose agar, and agglutinate antimeningococcus serum in as high a dilution as 1:200.

With the administration of antimeningococcus serum and the improvement of the patient, the cerebrospinal fluid changes become less distinct, the fluid becomes less turbid, the leukocytes diminish in number, the globulin decreases, and the bacteria become fewer and finally disappear entirely.

Other Suppurative Meningitis.—The fluid in pneumococcus meningitis, in streptococcus meningitis and in influenza meningitis differs but little from the fluid in meningococcus meningitis. Still there are some peculiarities characteristic of the special bacteria producing the disease. The fluid in pneumococcus meningitis, for instance, is creamy in consistency, has, as a rule, a pearly gray color, and on standing throws down a very heavy, often fibrinous, sediment. The fluid in streptococcus meningitis is turbid, but not as thick as either meningococcus or pneumococcus fluid. The respective bacteria are found in the direct smear and in culture (Fig. 41).

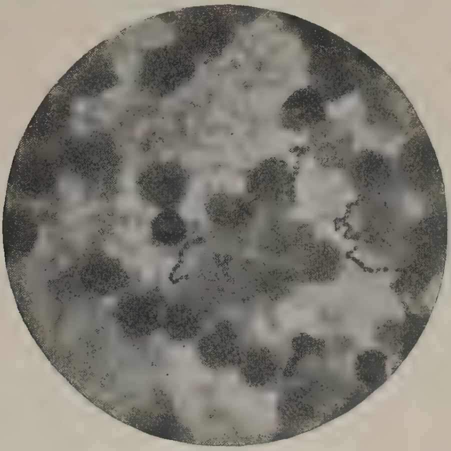

Fig. 41.—Photomicrograph showing streptococci in cerebrospinal fluid.

Colon Meningitis.—This rare form of meningitis is seen mainly in the newborn. It is seemingly caused by an infection reaching the meninges from the mucous membrane of the mouth. The cerebrospinal fluid is grayish yellow or brownish in color. The globulin and the cells are increased, the cells consisting mainly of polymorphonuclear leukocytes. The direct smear usually shows a short Gram negative bacillus, which acidifies milk and ferments all types of sugar.

SPINAL AND VENTRICULAR PUNCTURE

Historical.—As far back as 1842, cerebrospinal fluid was removed by Magendie from living animals by means of a fistula. From human beings cerebrospinal fluid was obtained, until a few years ago, only postmortem or after spontaneous outflow of the fluid following some trauma or disease of the cranial cavity. The first spinal puncture in a living human body was done in 1885 by Corning, who injected cocaine hypochlorate into the spinal cavity. The first ventricular puncture, as far as we could ascertain, was done by Middeldorf in 1886. Wynter in 1891, by introducing a tube into the lumbar region, drained the cerebrospinal fluid in cases of tuberculous meningitis. The perfection of spinal puncture, however, belongs to Quincke, who in 1891 described the method in all its details.

SPINAL PUNCTURE

At present, three routes are used to withdraw cerebrospinal fluid from a human being: (1) spinal, (2) ventricular, (3) occipito-atlas or cistern. Of these three routes, the spinal is the simplest and most advisable. Next to it is the ventricular route, and next to it, and the most dangerous route, is the occipito-atlas. Whenever possible, therefore, a spinal puncture should be done for the withdrawal of the cerebrospinal fluid from the body. Because of its importance, we shall discuss spinal puncture first.

Indications for Spinal Puncture.—Of late, it has been pointed out that the mere doing of a spinal puncture during the time a septic process is going on within the body may cause a meningitis, and one should, therefore, be guarded in the selection of cases for spinal puncture. We cannot subscribe to the view that spinal puncture when indicated is an unsafe procedure. We believe that not only is spinal puncture permissible, but it is imperative in many cases. The indications for spinal puncture are numerous, the following being a few of them:

1. Conditions showing symptoms of meningeal irritation.
2. Conditions pointing toward neurosyphilis.
3. Conditions pointing toward hemorrhage of the brain.
4. Conditions in which intraspinal injection is indicated.

Technic of Spinal Puncture.—While spinal puncture should be considered a regular operation, one can get along with only a needle and a few test tubes, if necessary. Of course, it is best to have not only the above-mentioned utensils, but also a manometer for measuring the pressure, a leukocyte pipette, a counting chamber, and various chemical reagents. The puncture itself, however, can be carried out with only a spinal puncture needle and a receptacle for the fluid. This makes spinal puncture a very accessible operation, which can be done at the patient's bedside.

The needle for spinal puncture in children should have a wide lumen. Otherwise, the thick fluid often encountered in suppurative meningitis may occlude it. There are several makes of spinal puncture needles on the market, most of them being 2.5 to 3 inches in length and 0.2 to 0.6 mm. in thickness. The needles are fitted with stylets. There are also needles with stopcocks made to fit various manometers for measuring the pressure of the fluid.

The test tubes for collecting the fluid should be small, sterile and chemically clean, otherwise the interpretation of the results obtained by examination may be misleading.

The preparation for spinal puncture should be the same as for any other operation. The needle should be boiled, the patient's skin should be washed with alcohol and iodine and the physician should scrub his hands and wear gloves during the puncture.

The puncture should be done with the patient on a table or cart, a bed not being steady enough for the work. The table should be low enough to allow the physician to sit down on a stool and still have free motion in his hands. All punctures should be performed with the patient lying down, preferably on his right side. The patient's body should be bent, so as to separate the intravertebral spaces. The needle should be introduced directly in the median line in very young children, and a few millimeters away from the median line in older children. The space of choice for the introduction of the needle is between the second and third interspace, although in children one may go one space higher without fear. A horizontal line drawn at the level of the crest of the ileum will usually strike the desired interspace.

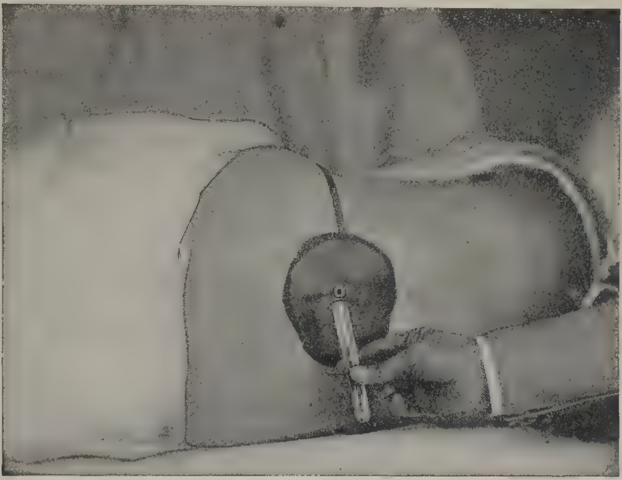

FIG. 41*a*.—Photograph of spinal puncture with needle *in situ*. The black line is one made at the level of the crest of the iliac bones, with an iodine swab as a landmark for the insertion of the needle.

After puncturing the skin, the needle should be directed forward and slightly upward, till a snap is felt, which usually indicates the puncturing of the dura. The stylet should then be removed and the fluid, if obtainable, be emptied into the test tubes. In case a manometer is used, the stopcock should be turned to prevent the escape of fluid, the manometer attached, and the pressure measured before the fluid is allowed to run into the test tubes.

It has been found best to use three or four test tubes for the collection of the fluid, so that if the patient moves around during the latter stage of the puncture and the fluid becomes bloody, as it often happens, the first or second tube could still be used for examination. It has also been found advisable to use the contents of either the first or the last tube for counting the cells in the fluid, so that a standard can be established for the number of cells in fluid; for it has been found that the cell content in the first few drops of fluid is higher than in the tenth or fifteenth cubic centimeter. After the desired amount of fluid has been obtained, the stylet should be re-introduced into the needle, and the needle pulled out from the patient's body. The wound should be closed with collodion.

After-treatment.—Rest in bed should be insisted upon after every spinal puncture. While it does not always prevent headache, if it does occur, the headache is not as severe as when the patient is up and around.

Failures in Spinal Puncture.—The two most important and most frequent failures are: (1) inability to obtain fluid, and (2) obtaining bloody fluid. While there are cases where there is very little or no fluid in the spinal canal, such as in marked atrophy of the spinal cord and in obstruction of the cord by a tumor, the average dry tap is due to the fact that the operator has not reached or has passed beyond the spinal canal. An idea of the measurement of the depth of the needle of the canal from the skin may help somewhat. In normal children, we found the depth by measuring the length of the needle after obtaining fluid, so that we were certain that the needle reached the canal. The result was as follows: In children under one year of age, the needle was found to have been inserted a distance of 2 to 2½ cm. In children between 1 and 2 years of age, the needle measured 2 to 3½ cm. In children between 2 and 4 years of age, the needle measured 2 to 4 cm. In children 4 to 7 years of age, the needle measured 2.8 to 4.0 cm., and in children 7 to 12 years, it measured 3.2 to 4.0 cm.

An experienced operator, however, needs no measurement of the needle to guide him in spinal puncture. He usually feels his way into the spinal canal.

If, in spite of several attempts, no fluid is obtained, the following may be tried, to make certain that the needle is in the spinal canal: the needle is left in place and a second needle is inserted one or two vertebral spaces higher. A normal salt solution is now injected slowly into the spinal canal through the upper needle. If the original needle has reached the canal, the salt will flow out through the needle. This procedure should, however, be carried out with great caution, as the second needle might be inserted too high and thus injure the spinal cord. In very fleshy children it is hard to feel the intervertebral spaces. Failure to obtain fluid is, therefore, more frequent than in slender children.

In deformities of the spine, due to rickets or tuberculosis of the spine, it may be impossible to reach the spinal canal. In such cases, it may be necessary either to introduce the needle obliquely to reach the spinal canal, or to resort to a ventricular puncture.

Obtaining bloody fluid is possibly the most frequent mishap in spinal puncture. It happens to both the inexperienced and the experienced. The blood is due to injury produced to the plexus of veins in the canal. Once blood is obtained the fluid cannot be used for cell count or for any chemical tests. It may, however, still be used for bacteriological purposes. A Wassermann test may be performed on bloody fluid, although at times the blood makes it anticomplementary. No new puncture for diagnostic purposes should be done for several days after blood has been obtained, as it takes three days for the blood to be absorbed from the spinal canal. However, when it is necessary to give intraspinal serum treatment, blood obtained on spinal puncture does not counterindicate the procedure.

Complications.—Death after spinal puncture has been reported in the literature. Death in those cases was seemingly due to the withdrawal of too great a quantity of fluid, thereby allowing parts of the brain to descend to the foramen magnum and become obstructed there. With the exception of a tumor of the cerebellum, no condition counterindicates spinal puncture, the possibility of death by puncture being minimal. We have done many punctures in cases of brain tumor without a single complicating death. Shock is not an infrequent complication. A hypodermic injection of atropine usually relieves it.

Injury to the aorta by spinal puncture has been reported, but is very uncommon. Breaking off the needle is not an infrequent complication in adults, but seldom occurs in children.

Headache after a spinal puncture is a common occurrence, but usually disappears in a day or two, especially if the patient stays in bed.

Radiating pain in the lower extremities takes place if filaments of nerves have been touched in the spinal canal by the needle. The pain, however, does not last long.

Edema of the skin in the lumbar region has been observed after repeated punctures. The edema, however, subsides in a few days.

If, for any reason, one is unable to obtain spinal fluid by the spinal route, and it becomes necessary to withdraw fluid for diagnostic purposes, the ventricular route must be resorted to.

VENTRICULAR PUNCTURE

Indications.—While ventricular puncture is not associated with any danger, it should not be performed unless lumbar puncture has failed repeatedly. Where the latter has failed either for the purpose of the removal of cerebrospinal fluid or for the introduction of serum, ventricular puncture should be performed without hesitation.

Technic.—All aseptic precautions should be taken for ventricular puncture that have been described for lumbar puncture. In addition to that, the hair around the anterior fontanelle should be shaven and alcohol and iodine applied. The operation proper varies with the age of the child. In infants the operation is very simple. In older children, in whom the anterior fontanelle is closed, the procedure is complicated and requires many surgical instruments.

In infants no anesthetic is necessary, while in older children it requires a general anesthetic. The technic in infants is as follows:

The patient is placed in the recumbent posture on a steady table, with the head at the end of the table. The head, shaven and washed with alcohol, is steadied by an assistant. A regular spinal puncture needle is now introduced a few millimeters to one side of the midline of the anterior fontanelle. The needle is directed forward and slightly downward to a depth of one to one and one-half inches (Fig. 42). After that, the stylet is removed and fluid is collected into one or more test tubes. If no fluid is obtained, the needle should be withdrawn and reintroduced, but should not be manipulated unduly while in the brain, in order to avoid damage to the brain tissue. If no fluid is obtained by the fontanelle route, and if the bones of the skull are still separated, the needle should be introduced between the frontal and parietal bones one to one and one-half inches deep. One must always keep in mind the danger of puncturing the medulla or other vital structures if the needle is allowed to go in too far.

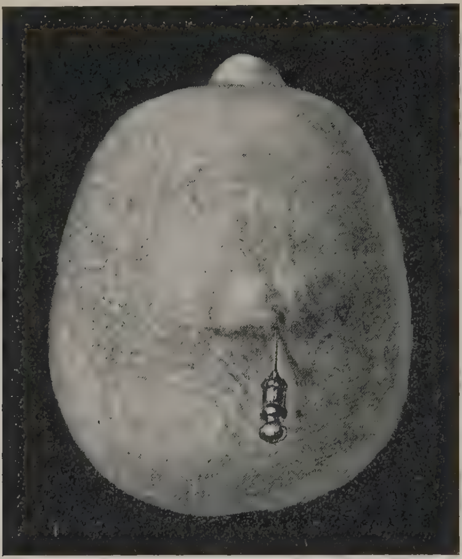

Fig. 42.—Photograph showing ventricular puncture on infant by way of anterior fontanelle.

CISTERN PUNCTURE

If both the spinal and ventricular routes have failed for the purpose of obtaining cerebrospinal fluid, or for the introduction of serum, cistern puncture may be resorted to. The technic, originally described by Wegeforth, Ayers and Essick, is as follows:

A spinal puncture needle is introduced in the midline of the back of the neck just above the spine of the axis. The glabella and the upper edge of the external auditory meatus may be used as a landmark for the insertion of the needle, for a plane passed through them to the back of the neck will pass also through the occipito-atlantoid ligament. In thin individuals a deep depression can be palpated between the occipital protuberance and the spine of the axis. This depression serves as another landmark. The needle is introduced at a depth of 3 to 5 cm., the average being 4 cm.

The preparation for the puncture and the collection of the fluid are the same as in spinal or ventricular punctures.

CHAPTER XV

ROENTGENOLOGY IN PEDIATRICS

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Introduction.—Roentgenology now occupies an important place in the practice of medicine and surgery, both as an aid in diagnosis and as a method of therapy.

Although a discussion of the discovery of the *x*-ray and the development of roentgenology would be of interest it seems wisest to refer to other texts for that, and likewise for the more technical side of the subject. The scope of this work is limited sharply to the diagnostic use of the *x*-ray in pediatrics, and that, too, to internal medical diagnosis. There are numerous works on the use of the *x*-ray in fracture, as well as in the special fields of the orthopedist, urologist, and *x*-ray therapist.

The internist and pediatrist have many problems in common, but, on the other hand, there are great differences in the two fields of work. The presence of gastric disorders, as represented by ulcers and neoplasms, is ever possible in the adult; it is the greatest rarity in the infant or child. Again, the picture of high blood pressure, atheromatous blood-vessels, hypertrophied heart, and renal changes—the cardiorenal complex—is to be anticipated in late adult life, but hardly to be expected in the infant. Congenital defects, congenital lues, scurvy, rickets, pyloric stenosis, Hirschsprung's disease, infantile tuberculosis illustrate the pediatrist's problems.

Aside from variations in disease processes, there are even differences in the anatomy, and in chemical composition, the younger organism containing more of water and less of mineral salts, while in older individuals healed processes often present areas of calcification and hence increased density. One would, therefore, anticipate peculiarities or variations in the roentgenological findings. It will be the aim to discuss these differences, or rather to emphasize the *x*-ray findings which are presented in the study of diseases of infants and children.

X-ray diagnosis of itself does not exist. The whole science of radiology is based upon the fact that the tissues of the body vary in the amount of resistance they offer to the passage of rays. For example: the bones are more opaque to the rays than the soft parts; lung tissue is more translucent than a calcified area in its midst. One is concerned at most with lights and shadows, the *x*-ray image being a shadowgraph. There are certain definite limitations, or even pitfalls, which must be recognized. Some of these may be enumerated:

1. The rays as employed in the routine work are divergent, hence the image is variously distorted, according to the position of the object with reference to the plate.

2. The plate or screen image represents the projection of a shadow or mass of shadows in a single plane. Allowance must be made for the superimposition of shadows.

3. Artefacts resulting from defects in screens or plates, or from the process of developing, must always be guarded against, and properly appraised when found.

4. The greatest pitfall of all is insufficient knowledge of anatomy and pathology. Interpretation of *x*-ray findings without these fundamentals would be as discreditable as the surgery of the novice without proper grounding in anatomy and pathology.

The literature of roentgenology is now enormous. It would be as impossible as futile in a work of this type to give a complete bibliography. Only such references are appended as would be of help in getting a general knowledge of the subject, especially as applied in the practice of pediatrics.

The Chest.—Roentgen or *x*-ray examination of the thorax in diseases of children has rapidly enhanced in value until at present its use is quite indispensable. This has resulted from better clinical and pathological information and a better technique. The latter is especially due to the perfection of apparatus and tubes to a point where good plates may be obtained by practically instantaneous exposure. Also, improved screens and fluoroscopic devices make fluoroscopy possible with a minimal current so that any reaonable exposure is free from danger of burns and injuries.

A warning may be interpolated here. *X*-ray examinations are simply a means to an end, an additional help, the findings to be interpreted as another symptom in the disease complex. This examination should never displace a most careful physical examination. The temptation is strong to save the time required for the physical examination, devoting a minute or two to the use of the screen. This leads to difficulties, for at best *x*-ray findings are simply studies of lights and shadows. One learns but little of the physical characteristics of the pathology producing the shadows, and worst of all a good clinical man may most rapidly deteriorate. Every-day practice in the methods of physical diagnosis is the price of clinical ability.

There is some question as to the relative merits of fluoroscope and plate. A choice of either is not necessary. Both are valuable, one cannot substitute for the other. The writer's method is to fluoroscope routinely every patient giving any suggestion of pathology in the thorax. Notes are made of the fluoroscopic findings and if these be positive a plate is made. The plate will show finer details overlooked by screening and it is further a permanent record of the condition at that date. Comparisons with subsequent roentgenograms give a true index of the course of the disease process. Moreover, the plate may disclose changes entirely overlooked in fluoroscopy, as, for example, early pulmonary tuberculosis of miliary type.

Fluoroscopy Technic.—An adjustable table is of great help. Infants are fluoroscoped, as a rule, in the horizontal position. It is hard enough to keep the patient in good position on the table; it is quite impossible to do so, holding a child up against a screen in the dark. A slight change of position greatly distorts all the relationships. A Coolidge tube is used for both screen and plate. It is quite important to have a shutter capable of being handled with one hand, the other hand being free for palpation and adjustment of position. Cumbersome harnesses for holding a struggling infant are not practical. Older children stand upright on an adjustable foot board. A little time spent in explaining to a child the process of the examination pays wonderfully. It prevents fright and promotes coöperation.

In screening, a general view of the entire chest first engages attention. One sees, in the sagittal or dorsoventral transillumination, two well-lighted areas, the lung fields. The fields are limited laterally by the curving rib shadows, below by the diaphragm. They gradually narrow toward the apex, conforming to the contour of the chest. The light areas, or lung fields, surround a dense middle shadow which is produced by the composite shadows of the spine, heart, great vessels, glands and other mediastinal tissues, and the sternum. Normal gland structures give no distinct shadows as such. With a suitable tube and a good screen a ribbon-like lighter area with sharply defined margins outlines the trachea. This may be traced from the neck down to its bifurcation between the third and fourth thoracic vertebræ.

It is now possible to observe in a general way the lighting of the lung fields, the size, position and movement of the heart shadow, the mediastinal shadow, the movement of the diaphragm, and the lighting up of the angles formed by the diaphragm and chest wall. Movements of the shadows within the chest cavity made visible by the screen offer a decided advantage over a fixed position registered by the plate. A comparison of the relative movements of the two halves of the diaphragm may at once give a clue to trouble in the chest, since the excursion of the diaphragm will be limited in the presence of inflammations such as pleurisy, pneumonia; or by the presence of an exudate, old adhesions or neoplasms. Forced inspiration, which may be complied with by older children, intensifies discrepancies in lighting, for under normal conditions the fields become much brighter, the diaphragm descends lower, and contrasts of lights and shadows stand out more distinctly on deep inspiration.

If there is defective lighting of any area, or limitation of motion in any part, the shutter should be cut down and minute study given to such areas. With a small lighting area one should now scrutinize the whole chest, the fields, the apices, the costo-phrenic angles, the hilus of the lung, the heart, and the area overlying the great vessels.

A careful study must be made of the lung fields. Though these are the lighter areas, the lighting is not homogeneous. One observes at the hilus or root of the lung rather dense shadows which branch out treelike into the lung, never quite reaching the periphery, and becoming less distinct as the pleura is approached. These shadows corresponding to the bronchial tree give the so-called mottled appearance of normal lung structure. At the root of the lung these radiating shadows become confluent and may be especially traced downward toward the diaphragm. They are more prominent on the right side, since those on the left are overlaid by the heart shadow. These correspond to the large bronchi. The cause of the shadows of normal lung structure has been much discussed. Some ascribe them to the bronchi, others to the vessels and lymphatics. A combination of these factors affords a simple explanation.

The area about the hilus or root of the lung also requires close study. Inflammatory changes, calcification and neoplasms produce changes in the bronchial structures, blood-vessels and glands. The shadows of these structures move but slightly or not at all with the movements of respiraton. Occasionally there are dense compact shadows, at some distance from the hilus, which move in a perpendicular plane with respiration, a proof of their location in the lung structure. Such shadows may be caused by calcification of some diseased area, most often a primary focus of tuberculosis. To demonstrate such shadows, especially if located near the base of the lung, will require the use of the tube at varying levels, because of the projection of the dome of the diaphragm up into the lung fields (Fig. 43).

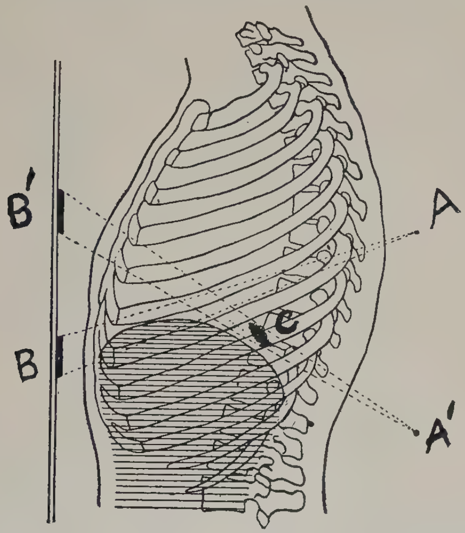

Fig. 43.—A and A′, Tube; B and B′, Screen or plate; C, Calcified area in the region of the posterior inferior border of the lung. From—Lehrbuch der Rontgenkunde, Rieder and Rosenthal. Barth, Leipzig, 1913.

Emphasis may here be placed on the fact that the shadows of the mediastinum, hilus, and lung structure in the infant or young child are much finer and less marked than in the adult. Even contrasts between bone and soft parts are less clearly cut. The greater density in the adult is due to a greater mineral content in the bones, atheromatous changes in vessel walls, thickening of bronchial walls from old repeated inflammatory processes, calcification of glands and cartilages, and the pneumoconiosis or anthracosis resulting from the inhalation of smoke or of dust arising in certain occupations. What might be considered as normal lung shadows in a man 40 years of age would betoken

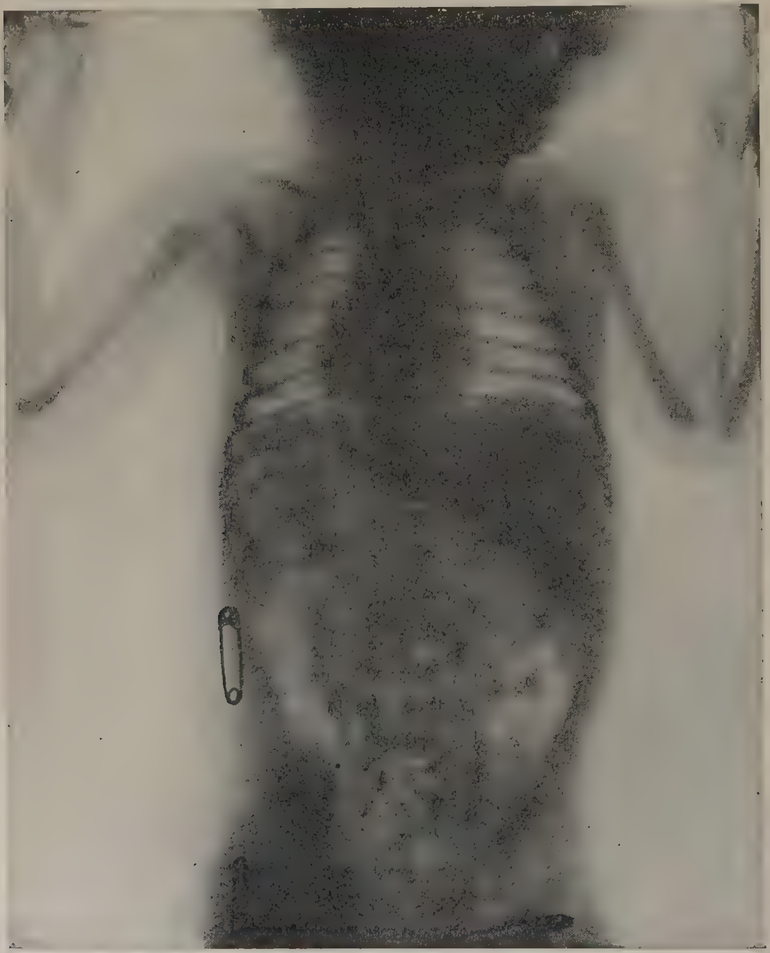

FIG. 44.—Plate of a newborn infant—normal. Note faint shadows thrown by the ribs, which run at right angle to the spine; the level of the diaphragm at the 7th rib; almost complete absence of hilus shadows and bronchial markings in the lung fields. There is a general indistinctness of the various shadows. This at first thought might be attributed to faulty technique—a poor plate. It is rather due to lack of contrast material in the infant's structures.

The plate also shows clearly the liver shadow. Because of the presence of gas in the bowels the intestinal coils are visible.

marked pathological changes in the infant or young child. It is necessary to reduce the adult normal scale in Roentgen studies when applied to pediatrics. There are also anatomical differences between the newborn and adult, as to height of diaphragm, the angle which the ribs make with the spine, and so on (Figs. 44–46).

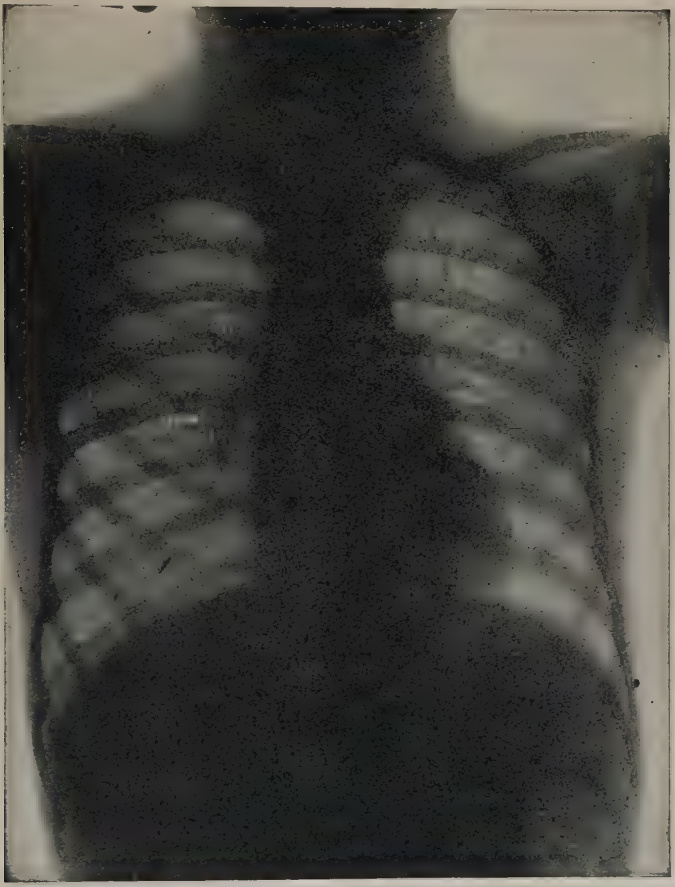

Fig. 45.—Plate of a normal child—eight years old. Note increased density of all the shadows, lower position of diaphragm, definite shadows at the root of the lung (1) and characteristic bronchial markings in the lung fields.

At this point a more detailed examination of the mediastinal and heart shadows may be made, though the order may be reversed and the lung examination made last.

One may begin with the heart, noting its location, position, shape, pulsations, changes in position with respiration and with change of the patient's posture. The cardiohepatic angle should be looked for as well as other evidence of adhesions between pericardium and diaphragmatic pleura. The size, shape and pulsations of the aorta also should be observed. Over the base of the heart one should look for the shadow of an enlarged thymus or any other abnormal shadow arising from the mediastinum.

After completing the dorsoventral examination the patient with arms elevated above the head, is rotated toward the left, bringing the right shoulder in proximity to the screen, the so-called right anterior oblique or semilateral position. This allows a lighting up of the post mediastinum which makes possible the investigation of enlarged glands, changes in the blood-vessels, and also permits of the study of a bolus descending in the esophagus. The spine stands out distinctly, permitting investigation of diseased conditions of the spine, such as an abscess

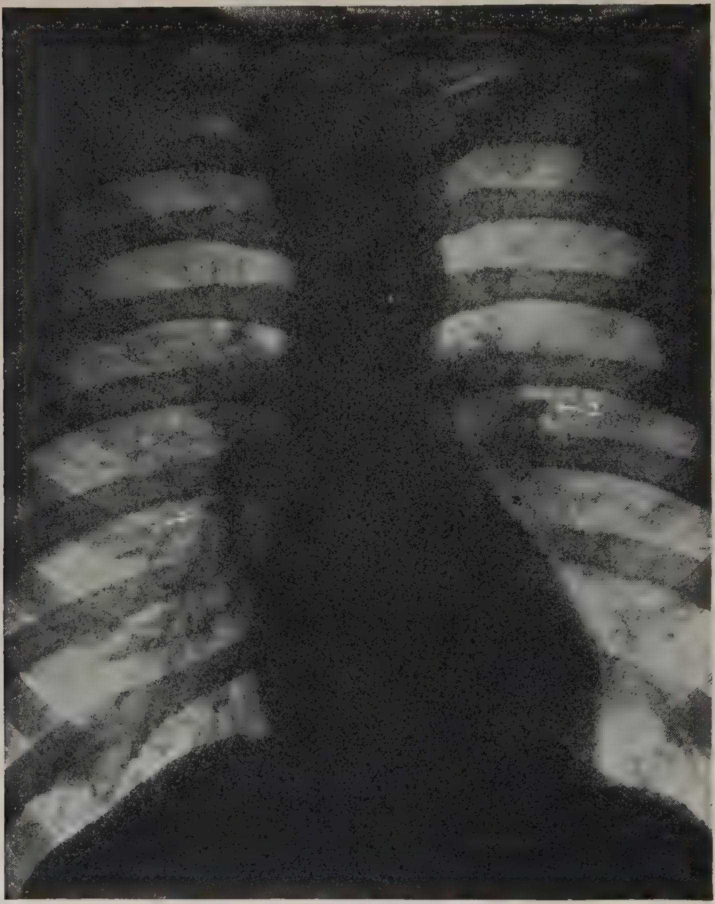

Fig. 46.—Plate of adult—normal. Note the contrasts. The ribs show definite osseous structure and project at an acute angle to the spine. There is a distinct hilus shadow (1) with areas of calcification, (2) bronchial tree shadows traceable well to the periphery—low standing diaphragm and distinct shadows of a blood-vessel (aorta) at the base of the heart (3).

of cervical Pott's disease. In the dorsoventral position the shadow of the heart and overlying structures prevents such a study.

The patient may now rotate until the back is in contact with the screen. This allows of another perspective of the chest contents, bringing the posterior portions of the ribs and spine into greater prominence, however. With arms now dropped to the sides, and the tube brought to such a level as to give perpendicular rays through the apices, the patient is requested to cough. This facilitates the observation of the lighting up of the apices. A contraction of the apical fields, or a disparity in the lighting of the two indicates some pathological change.

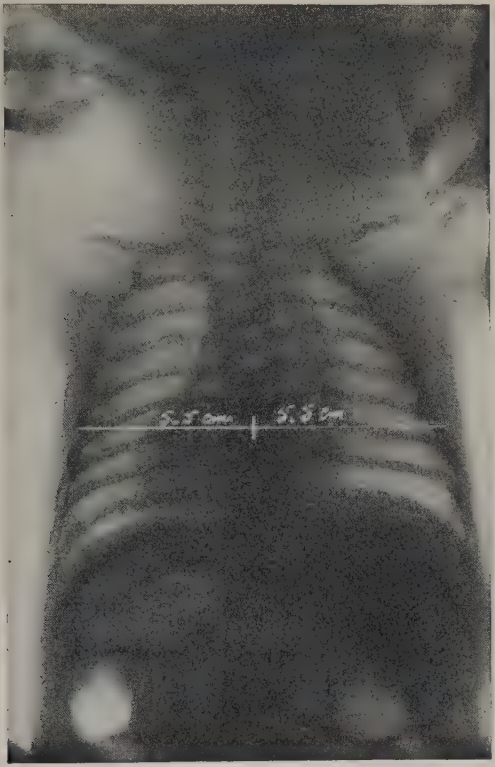

Fig. 47.—Plate of chest—giving normal relationships—measurement from middle of the spine to edge of ribs being equal on both sides—5.5 cm.

If positive findings are made with the screen, a plate or two plates for stereoscopic examination should be made. The writer professes no intimate knowledge of the technical side of the plate work but feels justified in suggesting two things:

1. In infants, the younger the child the more imperative, a soft tube is necessary. Calcification of bone is less than in the adult and a hard tube will wipe out all detail.

2. The patient must be flat and in extension. Partial rotation may throw the contents of the chest out of proper perspective. A normal chest under such conditions may give the impression of marked displacement of the heart, for instance. The shadows of other mediastinal structures may also be misinterpreted (Figs. 47, 48).

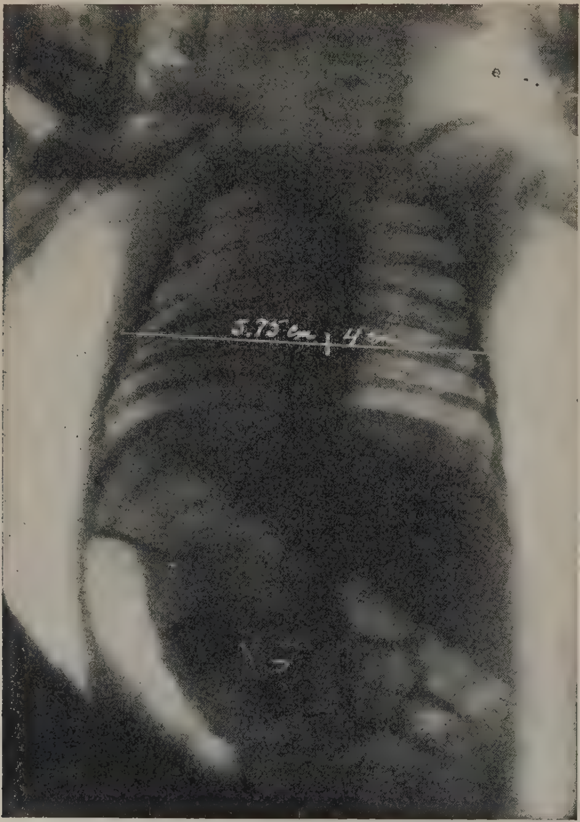

Fig. 48.—Same patient as Fig. 48. One of the two exposures made. At first sight one would think there was marked displacement of the heart and mediastinum to the left from some pathology within the chest. But this is due to faulty position of the patient in relation to the plate. See measurements from midspine to periphery.

For future study and comparison, data containing the name or serial number, date and character of the case should be attached to the plate.

Tuberculosis.—Tuberculosis in infancy and early childhood is protean in its manifestations, and a knowledge of the various *x*-ray images it may occasion will be of fundamental importance.

The pathology and course of tuberculosis in infancy is quite different from the types usually found in the adult. In fact we have learned from the studies made by Ghon, Albrecht, and Hamburg that tuberculosis in the adult is often the development of pathology definitely established in early life.

It may be stated that in 80 or 90 per cent. of the cases of tuberculosis, infection is aerogenous. In the remaining cases the most frequent nidus is the intestinal mucosa, and rarely the tonsils, teeth and nasal passages. By inhalation tubercle bacilli gain lodgment in the air cells of the lung structure, and here an acute pathological change is produced. This is the so-called primary focus. The disease may be arrested early and this area then becomes calcified. According to Sluka this may show up later in the *x*-ray examination as a small dense shadow, of at least 3 mm. diameter. It is located an appreciable distance from the hilus, usually near the pleural surfaces, and moves in a vertical direction with respiration. It is thus differentiated from a hilus structure.

However, before healing of a primary focus has been accomplished, tubercle bacilli may have been carried by lymphatics back to the glands at the hilus. The infected glands become congested, show hyperplasia and later undergo caseation. Here again the process may be arrested, the patient recover, and the glands undergo calcification. An *x*-ray examination in such instances will show shadows of the glands. This is the most frequent type of tuberculosis found in children.

Unfortunately the process may not be arrested, and this is the more likely the younger the child. Invasion of the lung, by continuity, from the hilus, produces, according to Sluka, a triangular area of infiltration—the so-called Sluka's triangle. Such shadows are seen, though not so frequently as Sluka's teaching would indicate. Again, the tuberculous change may be arrested with resorption, calcification and recovery.

At times infection of lung tissue may occur about a primary focus or by extension out from the hilus. A conglomeration of tubercles results, caseated areas break down, throwing infective materials into the bronchi and bronchioles. This results in a fan-like infiltration of the lungs, producing the tuberculous or caseous pneumonia. Or again, caseous tubercle-bearing materials may break into small blood-vessels, to be borne to all parts of the body, resulting in secondary and miliary types of tuberculosis.

Naturally the Roentgen findings will vary according to the type, location, and stage of the disease.

Primary Focus.—Fluoroscopically, its appearance has been described (Fig. 49).

Hilus Gland Tuberculosis.—The fluoroscope will show, in the earlier stages, rather indistinct shadows; later, when calcification has occurred, dense, sharply cut shadows. They lie at the root of the lung, usually extending out from the point of bifurcation of the trachea. Or one may see, in addition, a triangular shadow with the base over the gland shadows—Sluka's triangle. Dorsoventral illumination may reveal little, the gland shadows being also obscured by the

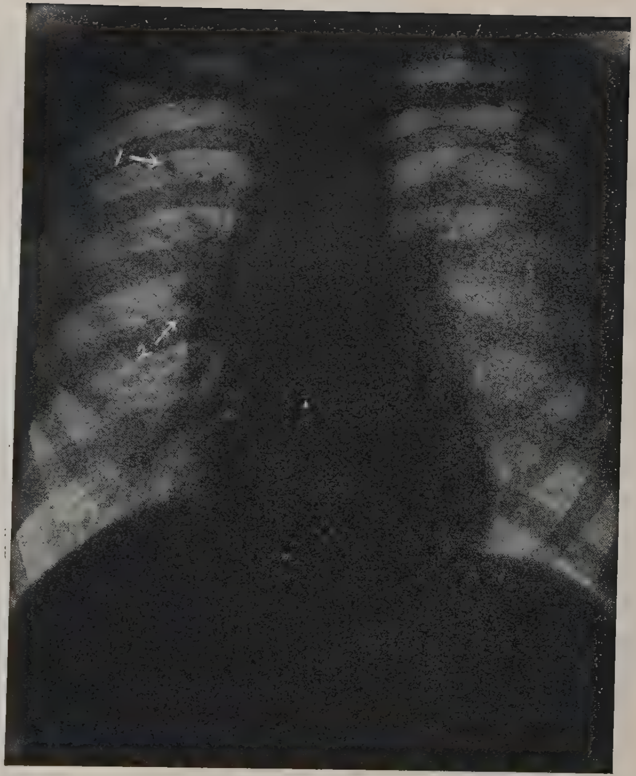

Fig. 49.—Primary Focus. This plate shows a dense, round, sharply circumscribed shadow well out in the lung field—a primary focus. (1) The regional hilus lymph glands produce definite shadows indicating a secondary tuberculous involvement (2).

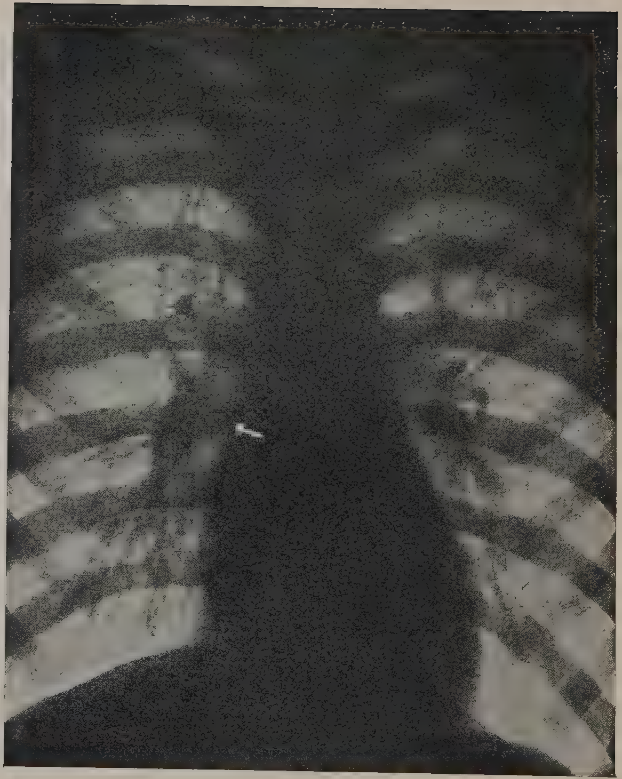

Fig. 50.—Hilus or bronchial glands. This shows gland shadows about the roots of the lungs. There is a general increase of density but also areas roughly globular in shape, outlining glands (1): there is a marked increase of bronchial shadows extending up toward the apices.

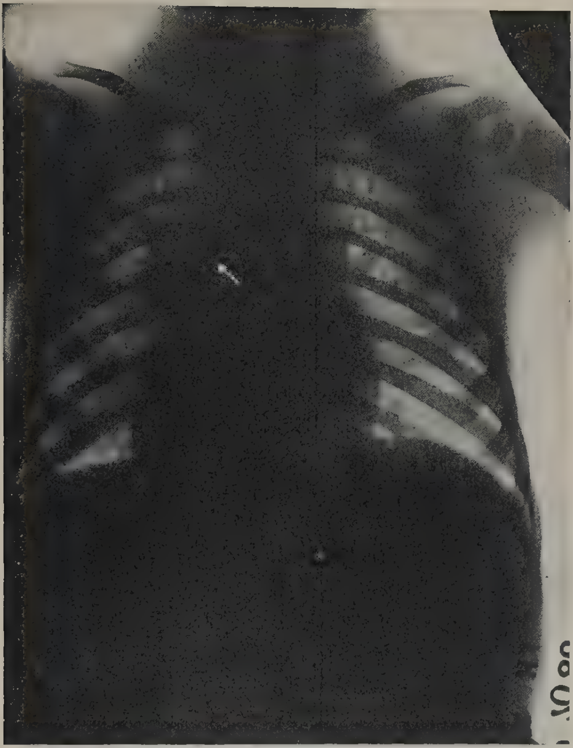

Fig. 51.—Hilus gland tuberculosis. Plate shows shadow of a mass of glands at the root of the lung. More pronounced on the left (1): bronchial tree shadows increased in both lung fields.

History and course: April 9th. J. H.—male 18 months—complaint, fever, cough—physical findings negative except for enlarged spleen and a positive D'Espine. April 18—von Pirquet positive—plate taken. April 30—exitus with typical symptoms and findings of a tuberculous meningitis.

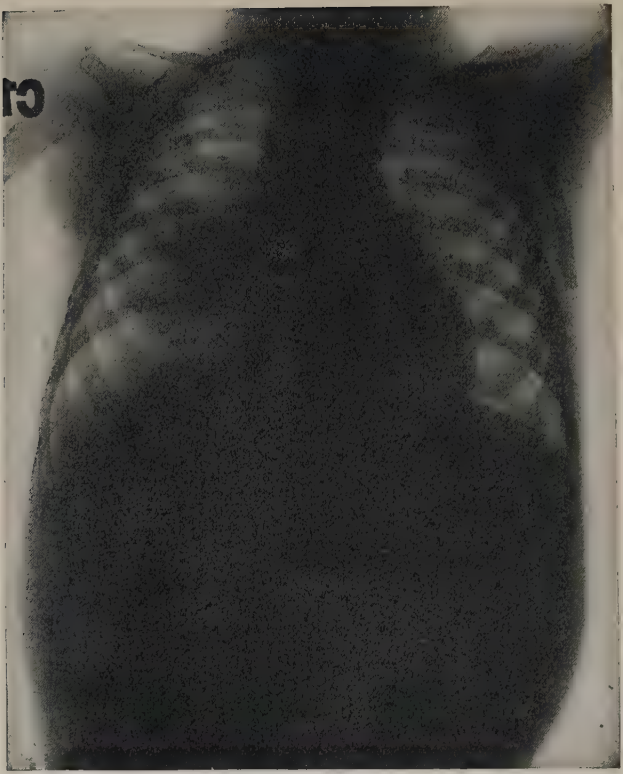

Fig. 52.—This plate shows the shadow of a gland or mass of glands at the right hilus. There is also the shadow of a surrounding infiltration roughly triangular in shape with base at the hilus and apex directed toward the periphery. This is a typical sluka triangle.

heart shadow. Plate studies may also fail to demonstrate them. Here the fluoroscope presents its distinct advantage for by using the right anterior oblique position, the posterior mediastinal space may be lighted up, and reveal a mass of glands. A careful search of the lung fields may reveal a shadow of the primary focus which will usually be found to have a regional relationship to the tuberculous glands. Plates permit of a study of finer details and provide a definite record of the case (Figs. 50–52).

Shadows of tuberculous glands and the Sluka triangle may vary from time to time. As healing occurs the shadow may decrease. On the other hand, after measles or pertussis the pathological process may flare up with a return of the shadow in greater density, and showing extension.

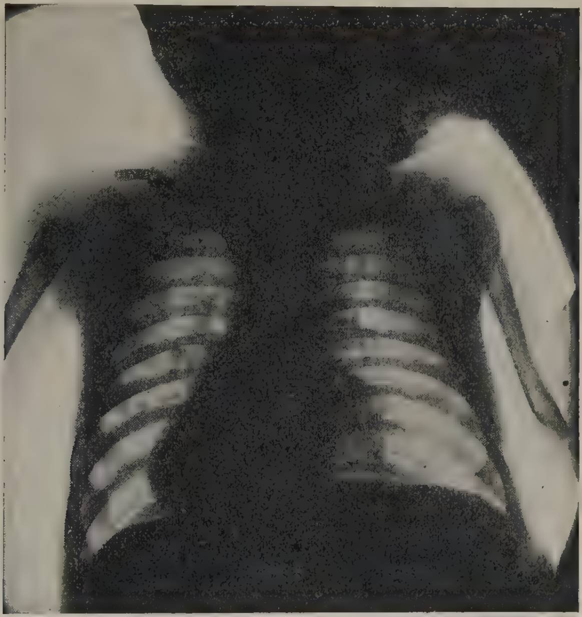

FIG. 53.—There is a very definite gland shadow at the right hilus suggestive of tuberculosis. This, however, was a case of bronchopneumonia complicating pertussis—the von Pirquet reaction was negative.

Here the limitations of the *x*-ray must be discussed. In the first place not every enlarged gland will cast a shadow. It must possess a certain density or, in other words, be in a certain stage of hyperplasia, caseation or calcification to produce a shadow. It is possible to have a rapid process revealing large soft glands at postmortem where the *x*-ray examination gives only indistinct, indefinite, non-characteristic shadows. On the other hand, it is most important to recognize the fact that many times hilus glands may undergo pathological changes such as to produce shadows, and still be non-tuberculous. The writer many times erred in his early enthusiasm for diagnosing hilus

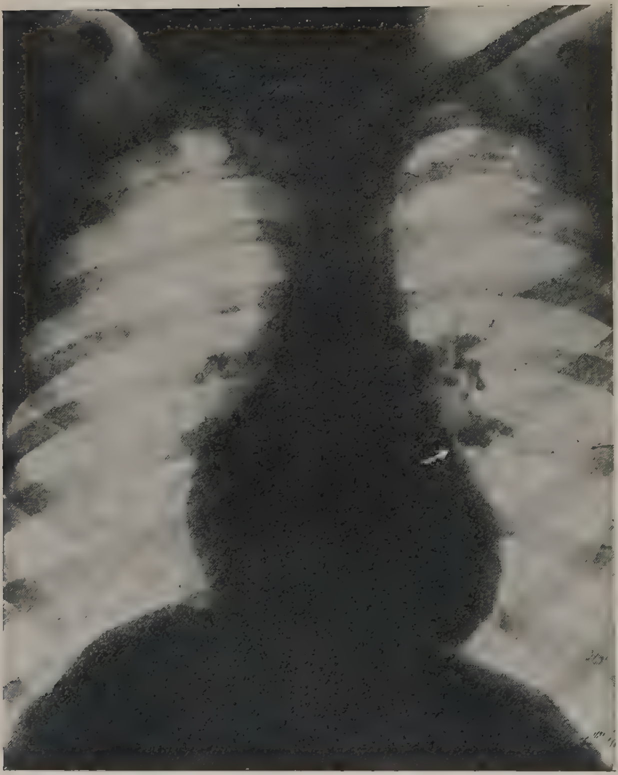

Fig. 54.—Definite gland shows—with very sharply outlined margins—well advanced calcification—negative tuberculin reactions.

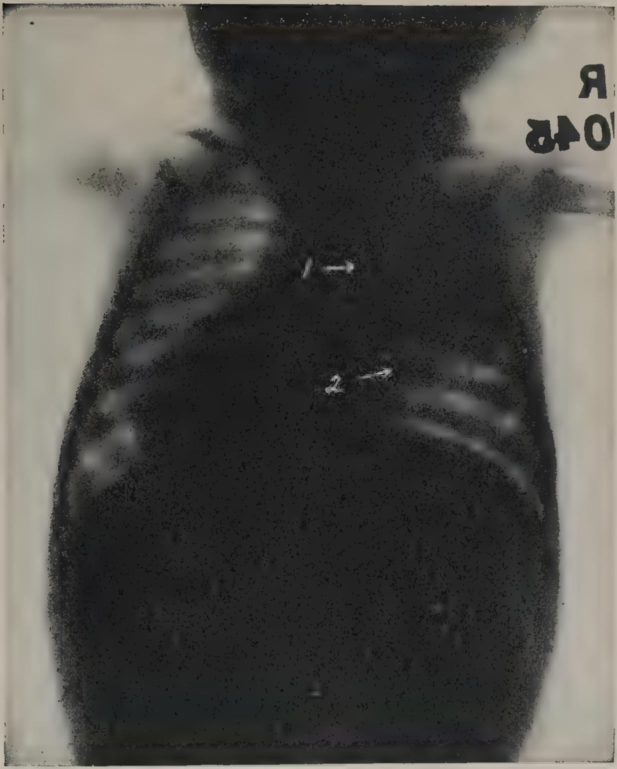

FIG. 55.—Tuberculous pneumonia. The plate shows a dense shadow in the right upper lobe (1) below gland shadows at the hilus (2) and moderate infiltration of the lung field. History of exposure to tuberculosis and positive tuberculin reactions.

gland tuberculosis. Subacute and chronic upper respiratory infections will produce inflammatory and hyperplastic changes resulting in an increase of density sufficient to produce distinct shadows. In such a case, a careful history, especially as regards tuberculosis, and the outcome of the tuberculin reactions, will help to differentiate the tuberculous from the non-tuberculous lesions (Figs. 53, 54).

Tuberculous Pneumonia.—Tuberculous infiltration of the lungs will produce a shadow very similar to pneumonia (see Pneumonia). However, one seldom fails to find a very definite shadow of the hilus glands draining the involved area. History, mode of onset, blood studies and the tuberculin reactions will serve to differentiate from pneumonia (Figs. 55, 56).

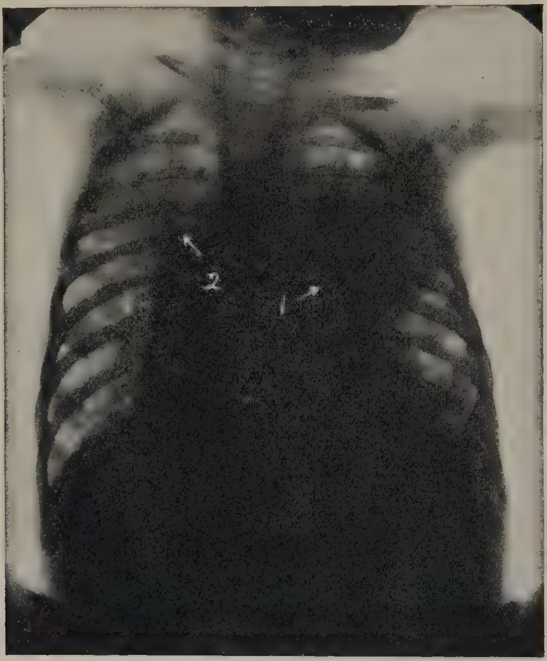

Fig. 56.—Tuberculous pneumonia. Note the shadow of consolidation in the right middle lobe (1) and smaller shadows in the left upper lobe—compare with plate of bronchopneumonia (Fig. 76).

History and course: Baby F—male—six months. Father in an advanced stage of tuberculosis—child coughing—losing rapidly in weight—consolidation over the right chest—von Pirquet positive—exitus 14 days later.

Miliary Tuberculosis.—Here an x-ray examination may be the only method of positive diagnosis. Physical signs may be entirely disproportionate to the gravity of the disease. A rapid loss of weight, fever, and râles in the chest may represent all the findings. On percussion the note is often hyper-resonant rather than dull. Further, tuberculin reactions may be misleading, for by experiment the writer has demonstrated in a series of cases that the same patient that formerly gave very positive von Pirquet reactions may fail to react anywhere from 10 days to 3 days before exitus. The fluoroscope may give entirely negative findings, or merely an impression that the lighting of the fields is not typical. The plate, however, is most characteristic. One sees a fine mottling of the entire lung fields, extending out into the periphery even up to the pleural surfaces. It has been described as a "snow flaky" appearance in the lung fields (Fig. 57).

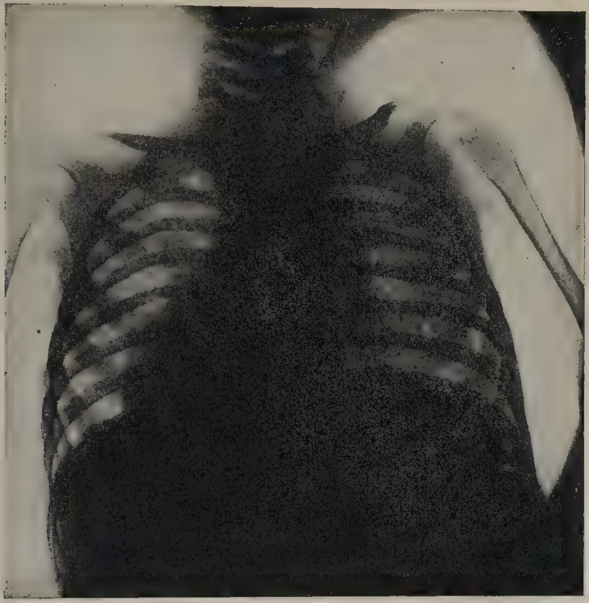

Fig. 57.—Miliary tuberculosis. Plate shows the typical "snowflake" mottling extending out to the very periphery-gland shadows at the right hilus.

Clinical history: Female infant 16 months old—delayed convalescence from measles six months before with cervical adenitis—exposure to tuberculosis in the home—cough and rapid loss of weight for the previous two weeks. Except for fever, emaciation, a positive D'Espine and râles in the chest physical signs were negative at the time the plate was taken. Von Pirquet at this time negative—previously it had been positive.

It was thought that this type of plate was pathognomonic of miliary tuberculosis, and doubtless it is if one insists on finding the snow flake shadows extending up to the very periphery and quite evenly distributed. But one can find a rather typical mottling along the bronchial tree, extending well out to the mid-lung field in chronic bronchitis and asthma cases; also, in metastatic malignancy and pneumoconiosis, which, however, are rare in pediatric practice (Fig. 58). Thus, one may accept a typical plate of this kind as pathognomonic of tuberculosis but must recognize again a limitation of x-ray methods. Failure to secure such a plate does not rule out miliary tuberculosis. Just as with hilus glands, we find that the tubercles must possess a certain density to give a shadow. Hence, very young

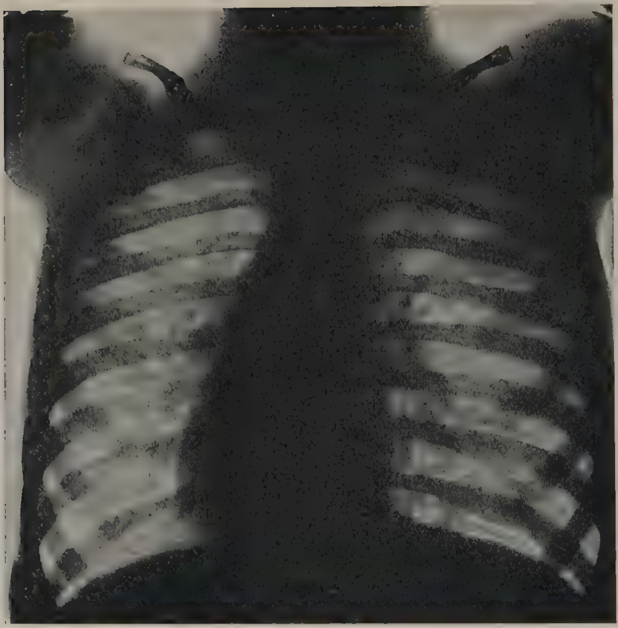

FIG. 58.—Chronic bronchitis. Plate shows an increase of the hilus shadows and nodular shadows along the bronchial tree. Compare with plates illustrating miliary tuberculosis. Note however, that the "snowflaky" appearance is limited to the central portions of the lung fields—while the periphery is clear. Plate was taken during an acute febrile attack accompanied with severe coughing and asthmatic type of breathing.

History: D. C. age four years—long standing history of repeated attacks of "cough and asthma" usually attended with fever.

Physical findings—râles, no signs of consolidation—von Pirquet negative.

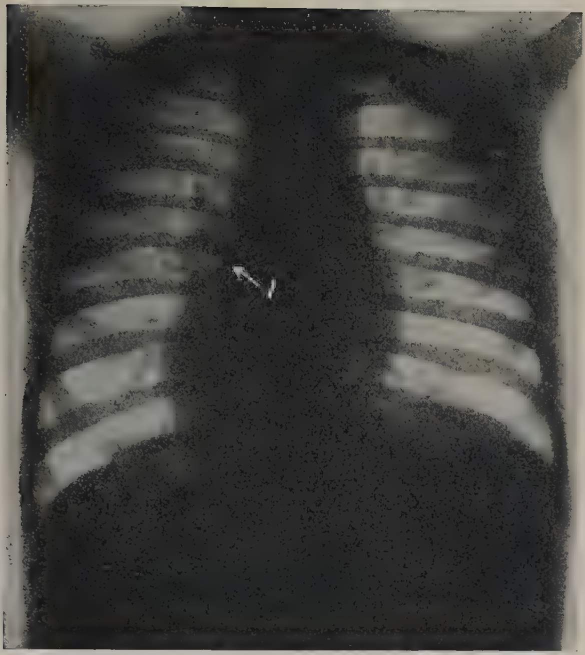

FIG. 59.—Miliary tuberculosis. Note: Fig. 59, 60, 61, and 62 are from the same patient—taken on the dates given. M. F. This plate (8/26/20) shows a large hilus shadow projecting beyond the heart shadow (1) to the left, increase of bronchial tree shadows in the left lung especially toward the apex.

History: M. F. age two years, father has open pulmonary tuberculosis. Has had slight fever for a few days and has been hyperirritable. Well nourished. Von Pirquet positive. Physical examination of the chest negative.

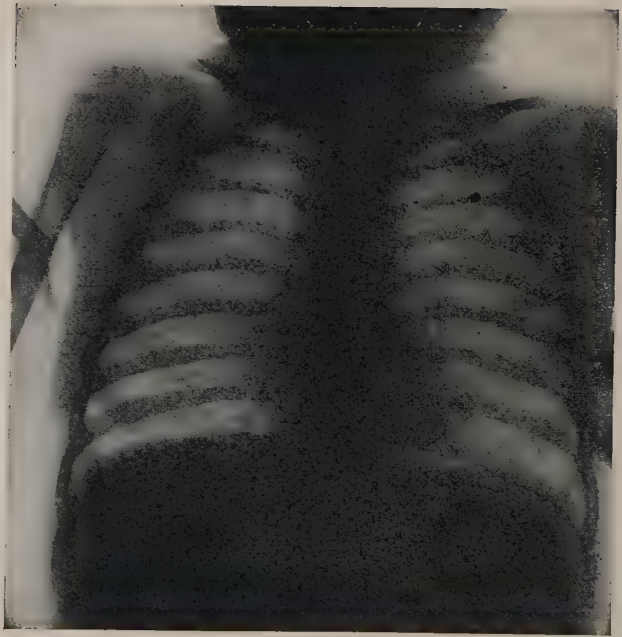

Fig. 60.—Miliary tuberculosis. This plate (9/5/20) shows the hilus shadow as before and in addition defective lighting of the lung fields—the tubercles being young lack sufficient density to cast a definite shadow.

History: Continued fever, meningismus, anorexia, hyperirritable. Von Pirquet positive.

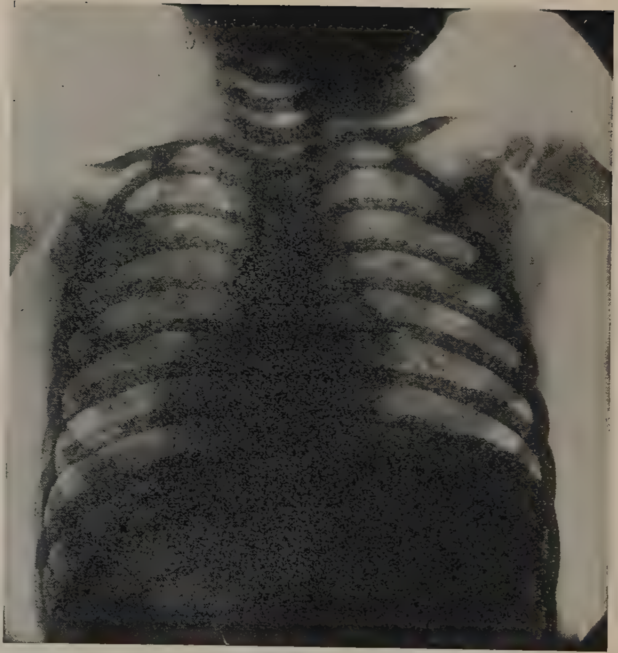

Fig. 61.—Miliary tuberculosis. M. F. (9/11/20). The lung fields show more distinct mottling—some tubercles produce definite shadows.

Course: Symptoms as before except for remission of temperature—still no cough and no definite physical signs within the chest. Von Pirquet positive.

tubercles will fail to give the usual findings. However, the progress of a case of miliary tuberculosis is rather rapid. Marked changes may be registered by the plate from time to time (Figs. 59–62).

Chronic Tuberculosis.—Chronic tuberculosis occurs in children, but much more rarely than in adults. The findings are similar to those in adults. Fluoroscopy may show a contraction of the apical fields with respectively poorer lighting of the involved apex. In the lung fields one notes a general thickening of the bronchial markings. Bead-like shadows may appear along the bronchial tree with fan-like infiltration of lung tissue. Later stages may show stippling out in the lung tissue very like the tubercle shadows of miliary tuberculosis. Conglomerations of such shadows with cavity formation may occur in advanced cases (Fig. 63).

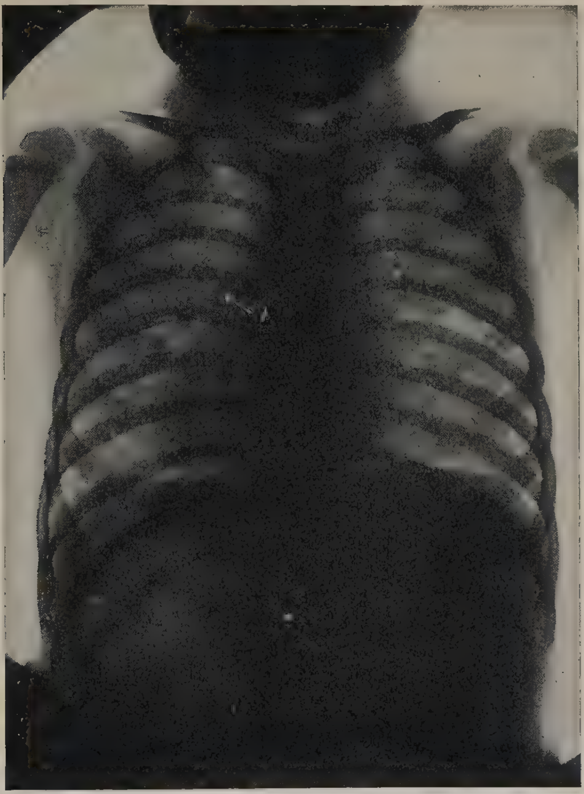

Fig. 62.—Miliary tuberculosis. M. F. (9/23/20). Typical plate of miliary tuberculosis—with areas of consolidation of lung tissue (1).

History: Progressive emaciation—no cough—fever—exitus three days later. On physical examination râles, areas of coarse or bronchovesicular breathing. At this date von Pirquet was negative.

Chronic fibroid phthisis in children is most apt to involve the pleura with resultant adhesions. Retractions may show lateral displacement of the mediastinum, heart, and in a good plate the trachea may show deviation from the normal course toward the diseased side (Fig. 64).

The "spitzen catarrh" or apical tuberculosis in childhood the writer thinks is a rarity, involvement of the apices being secondary to a tuberculous process in other portions of the lung. Cavity formation may occur in infancy and childhood, though less often than in the adult. There are no features peculiar to the youthful type. The fluoroscope may show lack of motion on the involved side, the hilus glands enlarged, with a shadow of demarkation about the cavity roughly circular in shape. The cavity if large may show fluid and air. The plate will confirm the same findings.

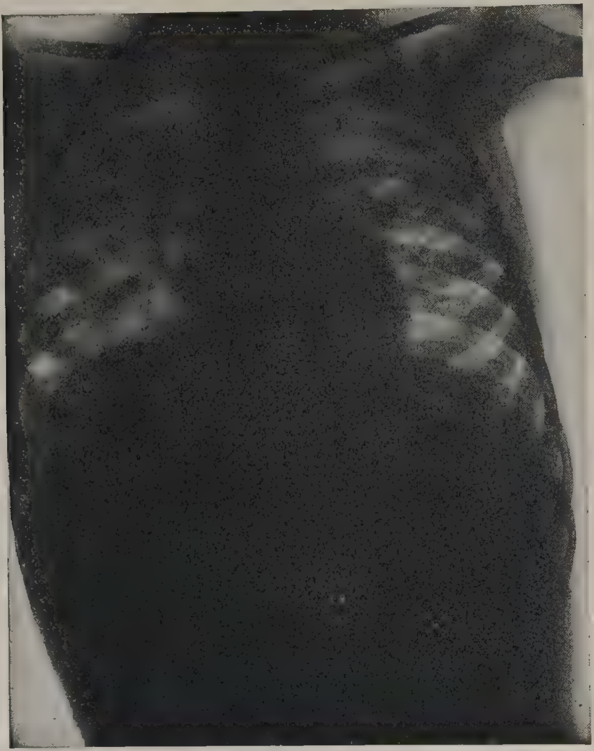

FIG. 63.—Chronic infantile tuberculosis. Disseminated shadows of miliary type over the right lower base—peribronchial thickening over the left chest —conglomerate shadows and areas of consolidation over the right upper chest.

History: Male—four years of age—ill for many months—entered hospital with complaint of a chronic diarrhea.

Findings: Extremely emaciated (has always been so) weighing 22 lb.—mild fever—over chest few signs, out of all proportion to the plate findings—abdomen level with ribs and having a doughy feel. Von Pirquet negative—Mantoux positive.

Tuberculosis of the pleura will be discussed under Diseases of the Pleura.

Lobar Pneumonia.—In pediatrics roentgenology is extremely valuable in the study of lobar pneumonia and its complications. The employment of the usual methods of physical diagnosis in this condition in infancy leaves much to be desired for these reasons:

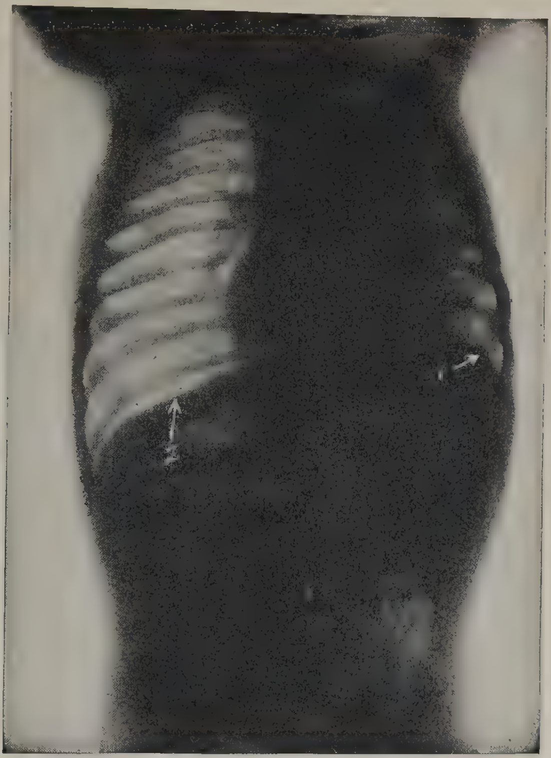

FIG. 64.—Chronic fibroid phthisis. The plate shows a shadow in the right chest—marked retraction of the heart and mediastinum to the right—the costophrenic angle is well lighted (1). Note the left lung field is exceptionally well lighted with faint lung markings—a compensatory emphysema (2).

History: Female infant eight months of age—incomplete recovery from influenza six months previously—persistent cough—recently has had fever—exitus three months later following pertussis.

Physical signs of displacement of the heart, dullness and poor breath sounds over the right side—hyperresonance with exaggerated breath sounds over the left. Von Pirquet positive.

1. The percussion findings are modified by the hyper-resonant elastic chest, stomach tympany, large liver and relatively high position of the diaphragm.

2. Movements of the child produce muscle changes which make it extremely difficult to elicit fine differences in tone with percussion.

3. The young child cannot coöperate, so that except for periods of crying the examiner is deprived of the help of such signs as increased fremitus, bronchophony and pectoriloquy. Again pneumonia often fails to conform to the usual text book picture. Unusual types of the

disease prevail in early life. The *x*-ray is a great help in these difficulties. A rather extensive use of roentgenology in chest pathology seems to warrant the following statements:

(*a*) Pneumonia is frequently overlooked.

(*b*) Many cases of abortive pneumonia are diagnosed "grippe."

(*c*) A positive diagnosis of lobar pneumonia is often made only at the conclusion of the course of the disease.

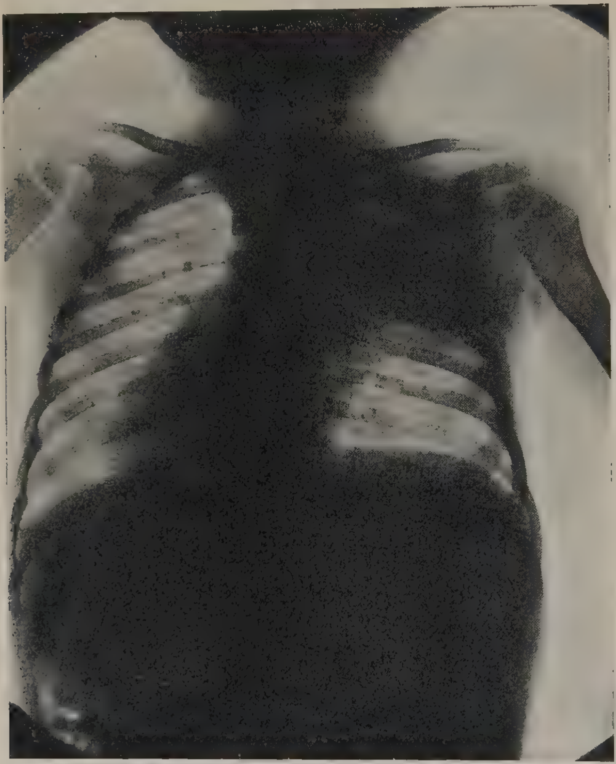

FIG. 65.—Lobar pneumonia. Plate shows a dense, homogeneous shadow with sharp, linear margin conforming to the anatomical boundary of the right upper lobe. History: Findings and clinical course typical for pneumonia.

(*d*) The plate often shows more extensive involvement of the lung than is suspected as a result of the physical examination.

(*e*) Incomplete and delayed resolution is often demonstrated by the *x*-ray when physical signs would indicate but little remaining consolidation.

(*f*) Parapneumonic empyema is often uncovered by *x*-ray examination when the physical signs would point to a simple consolidation of the lung.

The usual type of pneumonia may be readily recognized but the writer feels that *x*-ray studies and control during the course of the disease will greatly broaden our knowledge and in some instances will entirely change our conception of pneumonia and its complications.

The loss of air and the increased fluid and solid content (congestion, edema, and exudate) cause an increase of specific density of lung tissue in pneumonia. This reduces the permeability of such consolidated tissue to the rays, and shadows of greater or less density result. This is found to be true in a surprisingly short time after the onset of the disease, often hours or even days before signs obtained by percussion and auscultation would warrant the diagnosis of pneumonia. The fluoroscope shows the shadow and also the limitation of motion of chest and diaphragm. The plate registers the findings for future comparisons if needed. Further discussion of pneumonia will apply to plate studies.

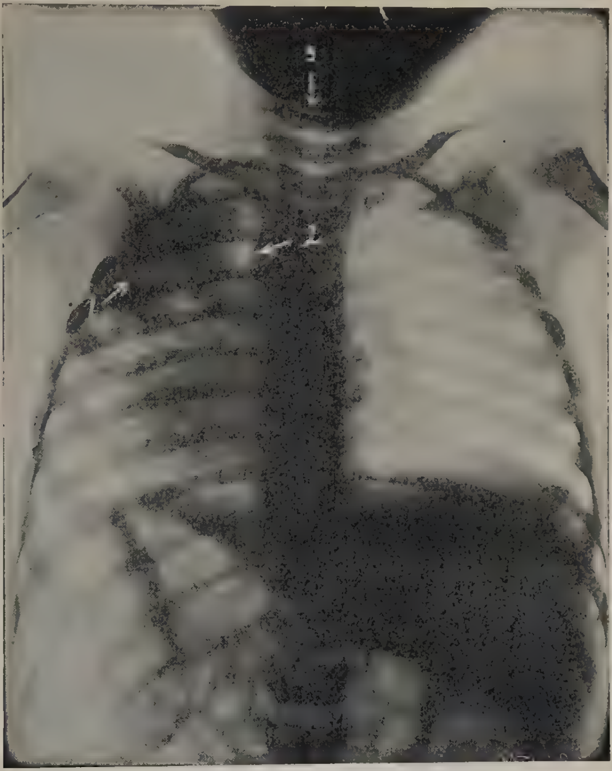

Fig. 66.—Pneumonia. A plate—shadow in the left upper lobe but not involving the entire lobe (1) peripheral—note air containing lung between the consolidated area and the root of the lung (2).

History, findings and course typical for pneumonia. Note outline and course of trachea (3).

The shadows of pneumonia have no specific characteristics though as a rule they are less homogeneous and dense than those of tumors and pleuritic effusions. On the other hand, they are more homogeneous than those of tuberculous lesions (excepting tuberculous pneumonia), bronchopneumonia and bronchiectasis. Further, pneumonic infiltrations occasion no displacement of mediastinal structures. The outline of the shadow may be sharply circumscribed and of characteristic shape if a single lobe is involved (Fig. 65).

The shadow may not involve a whole, single lobe. A part of one only may be consolidated (Fig. 66) or a portion of two lobes may cast a shadow. Again, the shadow may be peripheral, with lighted areas presenting between the root of the lung and the portion showing hepatization. It is the latter type of pneumonia in which physical signs may not be elicited and yet the Roentgen ray gives the necessary aid to correct diagnosis. For demonstration of the typical physical signs consolidation must be continuous from the trachea or larger bronchi to the periphery (Fig. 67).

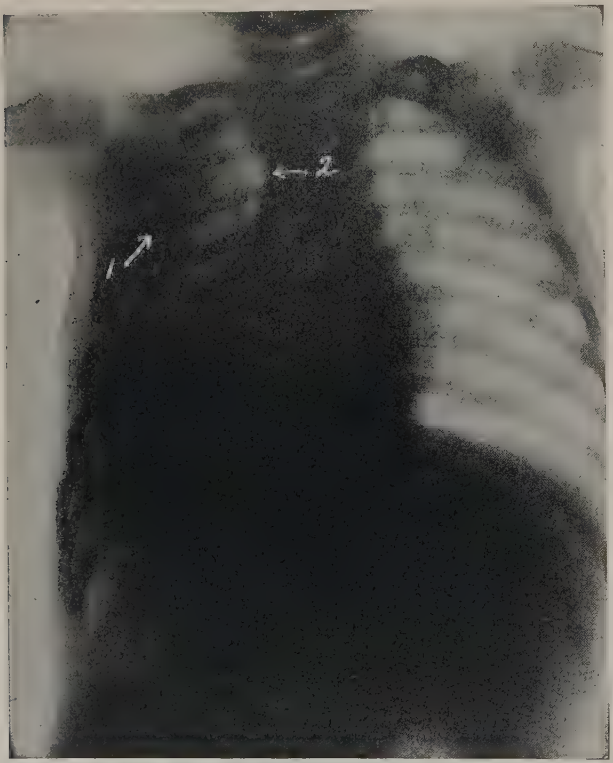

Fig. 67.—Lobar pneumonia. Plate—shadow involving the left lower lobe and a part only of the left upper. Note the consolidation in the periphery (1) with normal lung structure toward the hilus (2).

There has been much written about the existence or non-existence of central pneumonia. Though this question is of interest in a general way it is purely academic to roentgenology. Granting that the infec-

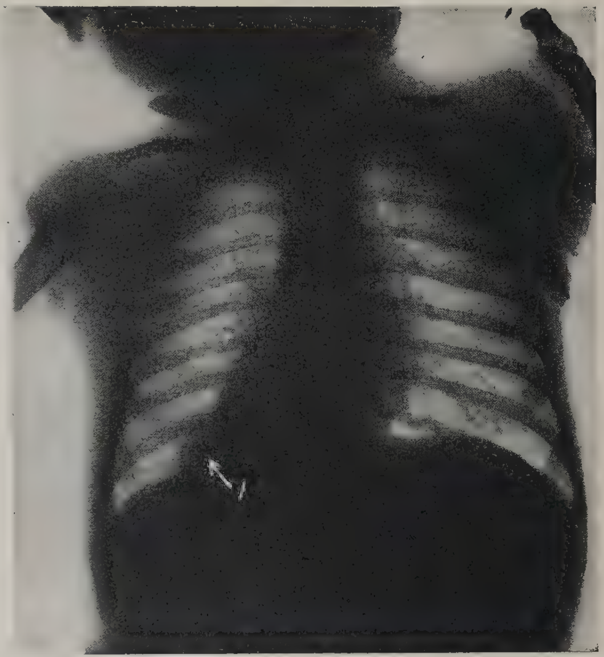

FIG. 68.—Central pneumonia (lobar). Note the shadow behind the apex of the heart (1). By oblique illumination with fluoroscope an extensive shadow was demonstrated back of the heart.

History and course that of pneumonia.

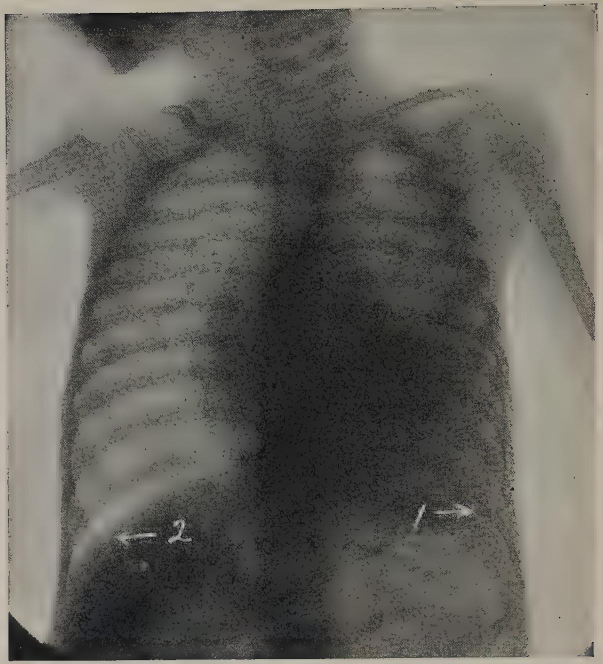

FIG. 69.—Lobar pneumonia. Pneumonic shadow involving almost the entire left lung—no displacement of mediastinal structures—obliteration of the left costophrenic angle (1)—compare with the normal (2).

History, findings and course typical for pneumonia.

tion takes place in the lung periphery and the consolidation proceeds toward the root of the lung, still the shadow may be central as viewed on the plate. To illustrate: The consolidation may begin in the periphery of the lung near the spine and proceed to the hilus. By anterior posterior lighting and the projection of the shadow on a single plane the shadow may appear central (Fig. 68).

We have demonstrated a shadow of pneumonia consolidation back of the heart by anterior oblique fluoroscopic examination, which we had failed to find by examination with the fluoroscope in dorsoventral illumination and by plate study.

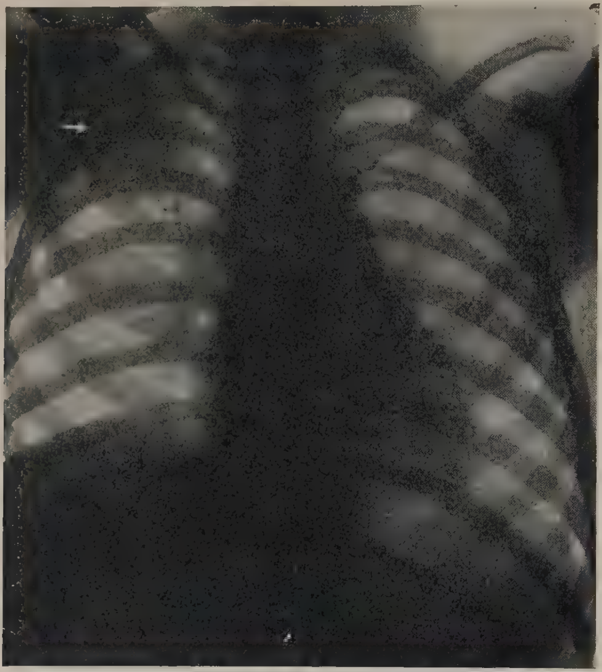

FIG. 70.—Abortive lobar pneumonia. Plate—(10/4/20). Shows definite shadow of consolidation in the right upper lobe—right diaphragm stands higher than the left.

History and course: J. S.—age five years—Oct. 2, awakened feeling ill—vomited at 8 a.m., cough and pain in the right chest—entered hospital at 8 p.m.—temperature 104—respiration 30—pulse 130—only physical signs diminished breath sounds. Oct. 3. At 9 a.m. felt well—temperature 99—respiration 22—pulse 90—no further signs in the chest—white count 32,000, 88 per cent. p. m.m. s. uneventful recovery. Plate taken Oct. 4. Courtesy of Dr. E. D. Anderson, Minneapolis.

The shadow of a pneumonic infiltration, if massive and involving an entire lung, may be homogeneous and of great density but without obliteration of the costophrenic angle as in effusion. But again in a simple pneumonia the costophrenic angle may be obliterated by a small serous, pleuritic exudate. This obtains in a much larger percentage of cases than is suspected. However, in an uncomplicated pneumonia the mediastinal shadows are never displaced (Fig. 69).

The shadows of pneumonia may vary greatly in density, due to two factors.

1. There is often a progressive involvement of a lobe which does not of necessity include the entire extent or depth of lung tissue.

2. Clearing of a lung often follows in the order in which consolidation appears, *i.e.*, in certain areas a lobe may be clearing while in others the disease is stationary or progressive. These developments are brought out by fluoroscopic examination with a narrow diaphragm opening and the tube shifted at varying perpendicular levels.

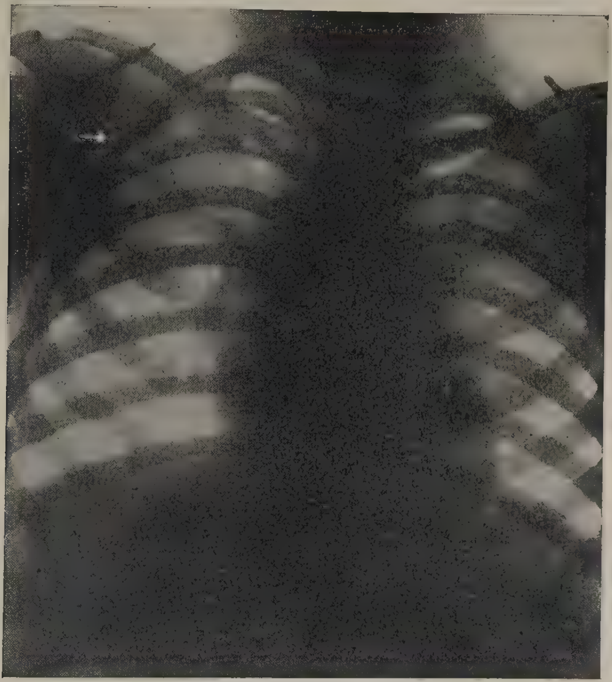

FIG. 71.—Abortive lobar pneumonia. Plate of same patient as shown in Fig. 68 taken three days later—note the shadow in the right upper lobe has entirely disappeared—both sides of the diaphragm at the same level. Courtesy of Dr. E. D. Anderson, Minneapolis.

Abortive Pneumonia.—Routine examination of the chest of children showing a sustained febrile course of more than 24 hours would give surprising results. Unquestionably there are abortive pneumonias lasting but a few hours and terminating by crisis. Not only is the shadow characteristic of pneumonia but onset, clinical course and laboratory findings are confirmatory. Such cases are not rare (Figs. 70, 71).

"Migratory" Pneumonia.—The writer has followed many cases with fluoroscope and x-ray plates and has been surprised at the fre-

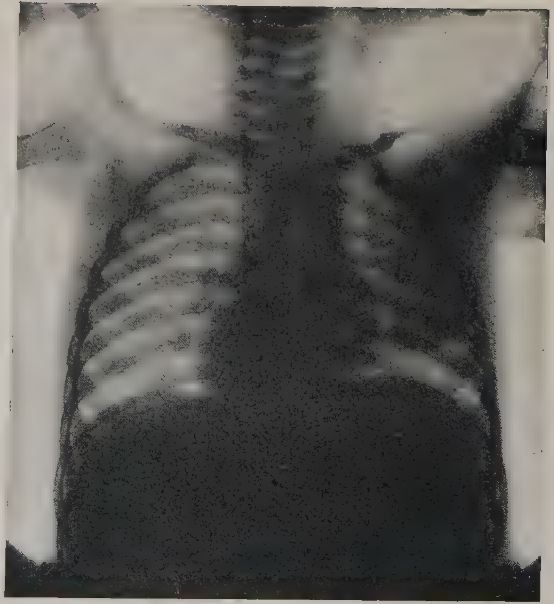

FIG. 72.—Migratory pneumonia. Figures 72–75 inclusive and plates of the same patient taken over a period of one month—see history notes.

Plate: Note the shadow in the right chest—apex and base of lung clear.

History and course: M. G.—female—age 15 months. Entered hospital (4/8)—with history and findings of pneumonia—temperature normal—white count 11,500—plate figure 30—taken (4/10)—von Pirquet negative.

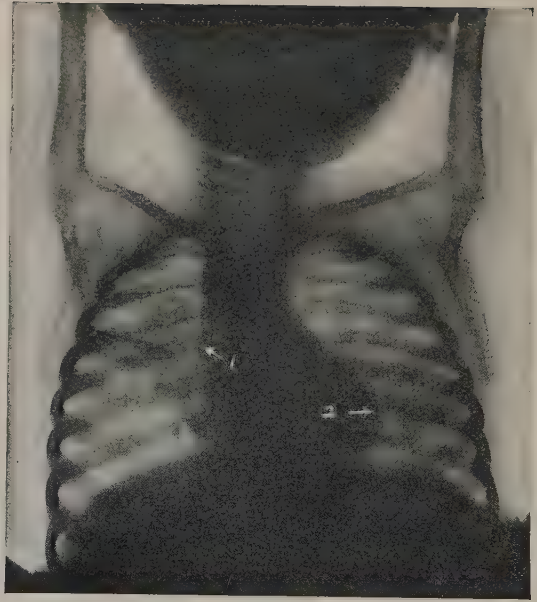

FIG. 73.—Plate shows clearing of the shadow on the right (1) and an extensive shadow on the left (2).

Physical finding of pneumonia on the left—temperature 104—(temperature had been normal for eight days) white count 39,500. Plate taken (4/21)—temperature curve reached normal (4/22).

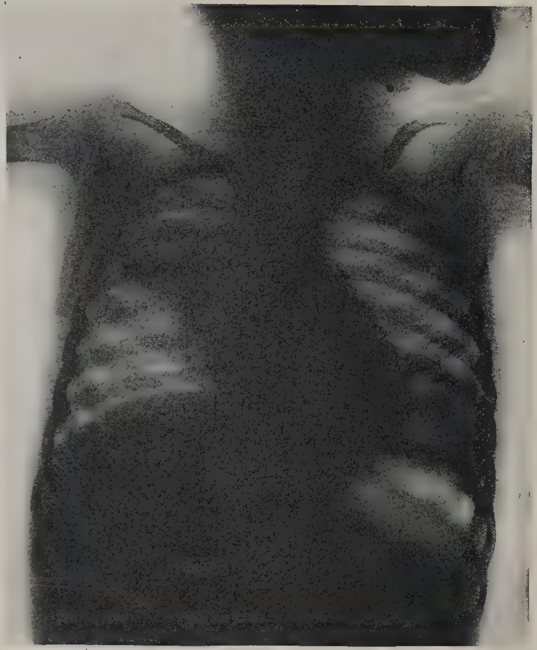

FIG. 74.—Plate shows shadows in a part of the right upper lobe and in the left lower lobe.

Course (4/29)—temperature suddenly rose to 104—white count 42,000—signs of pneumonia on both right and left sides. Plate taken (5/2).

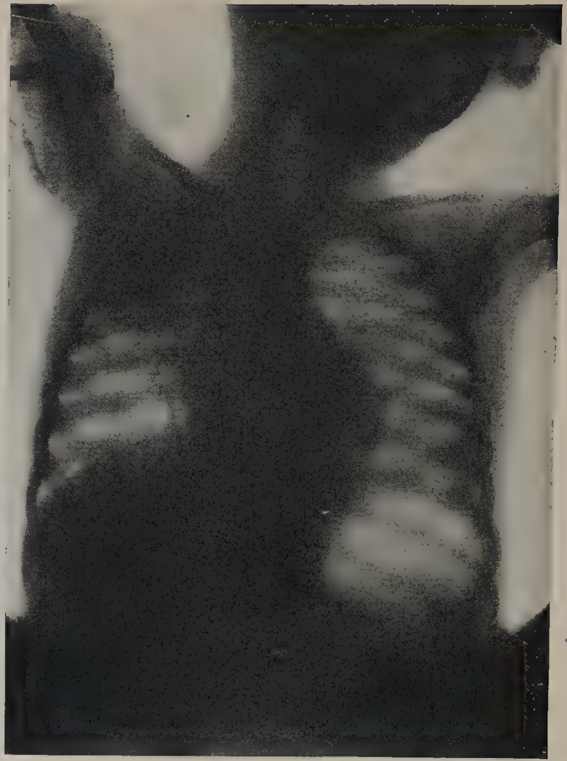

FIG. 75.—Plate shows extension of consolidation to the right apex—the rest of the lung fields have cleared. Plate taken (5/15).

quency with which shadows may appear and clear in various parts of the chest. Each new area involved is usually announced by a rapid rise in temperature, increased respiratory rate, leukocytosis and the signs of pneumonia. The shadows are not limited to contiguous parts of one lung but may involve different lobes or sides of the chest (Figs. 72–75).

Unresolved Pneumonia.—Usually, shortly after the crisis the lungs clear rapidly. Diminished breath sounds and râles may be present after the lung fields appear clear, but there may also be found some thickening of the bronchial tree, enlargement of regional glands and limitation of the diaphragmatic excursion or a peculiar lighting at the site of the consolidation. Often the findings are suggestive of tuberculosis. Discrimination must be exercised in the interpretation of these persistent shadows. They may result from a thickening of the pleura, a pleural effusion or incomplete resolution. It was formerly taught that an unresolved pneumonia usually meant tuberculosis. But such an outcome is a rarity considering the number of such cases. Doubtless one should be cautious as to prognosis, but the *x*-ray has shown that a tuberculous process in unresolved pneumonia in childhood is certainly not so frequent as was formerly supposed.

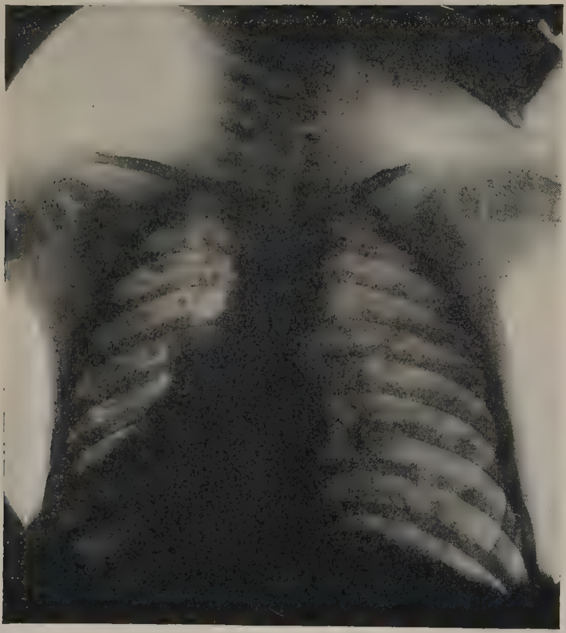

FIG. 76.—Bronchopneumonia. Plate—note the large hilus shadow and the diffuse mottling throughout both lung fields—compare with Figs. 55 to 63.

History: D. N. age 19 months—rhinitis for one week—entered hospital (4/29)—with temperature 103—pulse 160—respiration 70, extreme dyspnea. Physical signs, diminished breath sounds with râles of various types—white count 28,000. The von Pirquet was negative—complete recovery—discharged (5/25).

Bronchopneumonia or Lobular Pneumonia.—In this condition, which in children often follows grippe, influenza, measles, pertussis, and diphtheria, the *x*-ray may be of distinct aid though not with the sharp definition to be found in the lobar type. One may find small diffuse shadows extending out along the bronchial tree from hilus to periphery, but these shadows are not distinctive. Further, an increase of the hilus shadows may be demonstrated. The picture may be a counterpart of early tuberculosis (Fig. 76).

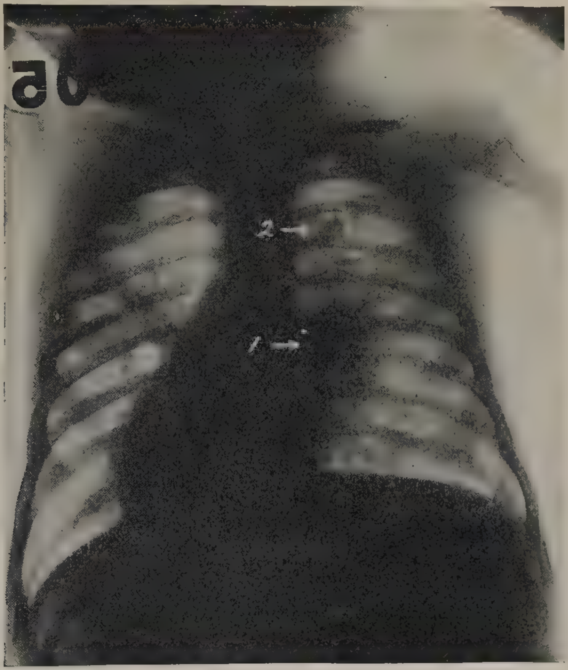

FIG. 77.—Bronchopneumonia. Plate—note extension hilus shadow on the right side (1) with diffuse increase of bronchial shadows, in some areas confluent (2).
History course: R. F. age 15 months—entered hospital (11/1/18) with the findings of bronchopneumonia—negative von Pirquet—died (11/8/18) plate taken on the day of death—autopsy report—bilateral bronchopneumonia.

Diagnosis of involvement of the lung may be made, but determination of the exact pathological condition must often rest on the history, blood findings, outcome of tuberculin reaction and subsequent course. On the other hand, the areas of infiltration and increased density may become confluent, producing a massive shadow of greater or less extent simulating a lobar pneumonia (Fig. 77).

As a rule, however, shadows are found in both lungs and there are multiple areas of increased density, hazy in outline, situated along the course of the larger bronchi.

A rather frequent type of bronchopneumonia, especially occurring in feeble infants, complicating pertussis and measles, shows a dense shadow on both sides of the spine extending a considerable distance out into the lung fields, the so-called paravertebral pneumonia. This should not be confused with tuberculosis which tends to be disseminated widely through the lung fields out toward the periphery.

Bronchopneumonia may resolve quickly or persist for days, weeks or months. If such a picture complicates measles or pertussis one must weigh the possibility of a superimposed tuberculous infection, or the lighting up of an old tuberculous focus. Roentgen study may not help, and worse still, a negative von Pirquet reaction does not rule out tuberculosis since a positive result of the reaction is often held in abeyance during the course of measles and pertussis and for some considerable time after. The clinical course is the criterion, and subsequent *x*-ray examinations will clearly show progression or regression of the disease.

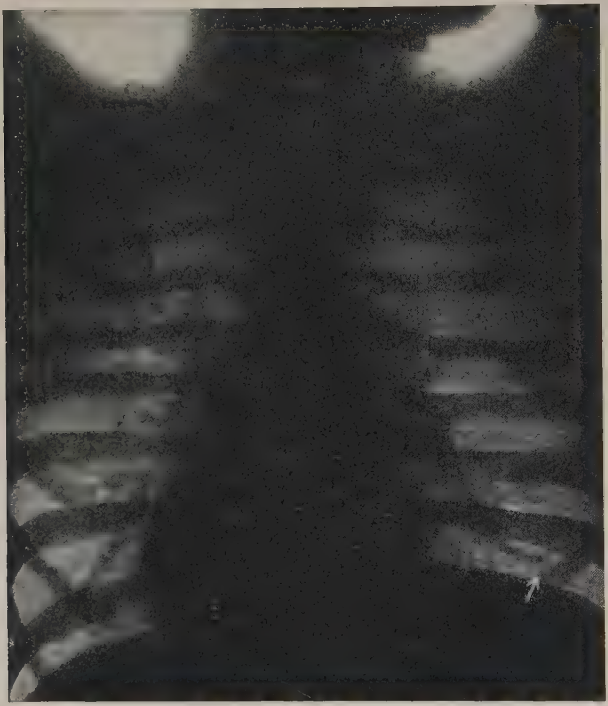

Fig. 78.—Asthma—chronic bronchitis. Plate—note diffuse hilus shadow—marked increased bronchial tree shadows, especially toward the base and nodular shadows along the course of bronchi (1).

History: O. N. 13 years of age—whooping-cough at seven years, chronic cough and "asthmatic attacks" since.

Physical findings—undernourished—anemic—transitory mucous râles in chest—von Pirquet negative.

Acute Bronchitis.—In this condition the *x*-ray is of little if any value. A surprisingly great amount of subjective symptoms, as fever, anorexia, cough, and abundance of râles of various types, will be accompanied by no changes in the lighting of the lungs as observed by screen or plate.

Chronic Bronchitis.—Chronic bronchitis may give very positive findings. These consist of thickening of the bronchial tree in the form of indistinct nodular shadows, almost an exact counterpart of the picture of miliary tuberculosis. The shadows may be found well out to the middle of the lung fields and accompanying a large hilus shadow. There is, however, no definite consolidation of lung tissue proper (Fig. 78). Also, influenza and pertussis may produce like shadows in the lung fields. This illustrates again that the mottled appearance of a plate is not pathognomonic for miliary tuberculosis. A careful history, painstaking physical findings and careful laboratory work may allow of a positive diagnosis, but even with all this, pitfalls may beset a too dogmatic x-ray diagnosis. The course of the disease and subsequent x-ray examination will serve to clarify the issue.

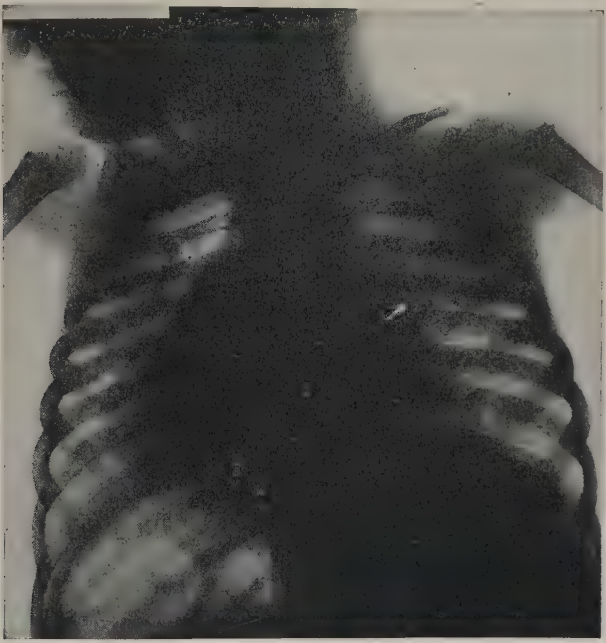

FIG. 79.—Bronchiectasis. Diffuse shadow at root of the lung—lighter areas presenting cross sectional view of dilated bronchi (1) peripheral lung fields clear.

History: A. J.—three years of age—cough for two years—with abundant expectoration characteristic of bronchiectasis. Physical findings negative except for rhinchi and mucous râles—von Pirquet negative.

Asthma.—The Roentgen examination in this condition is not of great practical value. During an acute asthmatic attack the fluoroscope may reveal the chest to be in the position of fairly complete inspiration, with little respiratory movement and limited motion of the diaphragm. In older children and in chronic cases of long standing the bronchial tree shadows are much increased, due to a persistent accompanying bronchitis. The picture is then one of chronic bronchitis plus emphysema (see Emphysema).

Bronchiectasis.—While abscess and phthisis cavities are rather infrequent in infancy and childhood, on the other hand, bronchiectasis is very frequently found. The pathology consists of enlargements along the bronchial tree, saccular or fusiform in shape. The usual causes are chronic cough accompanying pertussis, chronic bronchitis, and asthma. A peribronchitis, which is the usual accompaniment of pertussis, naturally predisposes to dilatation of the bronchi involved. Likewise spasmodic constriction of the bronchi with expulsive coughing in certain types of asthma is a frequent causative factor. Milder grades of bronchiectasis may show simply an accentuation of bronchial tree markings. This may be limited to one lung and is found much

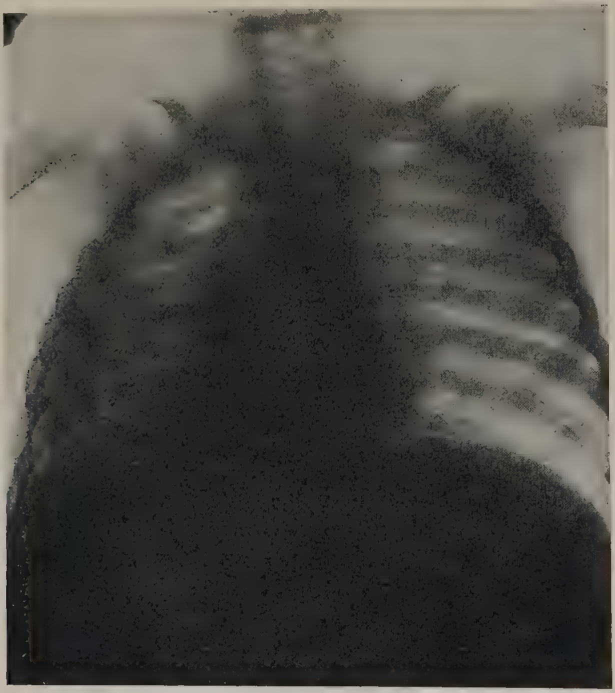

Fig. 80.—Bronchiectasis with pneumonia. Note homogeneous shadow over left lower lung—pneumonia.

History: Acute onset of fever, dyspnea and rapid pulse, leukocytosis and signs of pneumonia. This patient had repeated attacks of pneumonia complicating bronchiectasis—grew progressively worse and eventually died with the symptoms of pneumonia and acute heart dilatation.

more prominently toward the base. A severer grade often shows much denser shadows with nodular enlargements, areas of dilatation, and sacculation of the bronchi and collection of fluid (Fig. 79).

Very severe grades may be found, with both lungs involved and the process widely disseminated. The plate then resembles a bronchopneumonia, or even a lobar pneumonia during resolution. Such patients often run the clinical course of a pneumonic infiltration in the bronchiectatic field (Fig. 80).

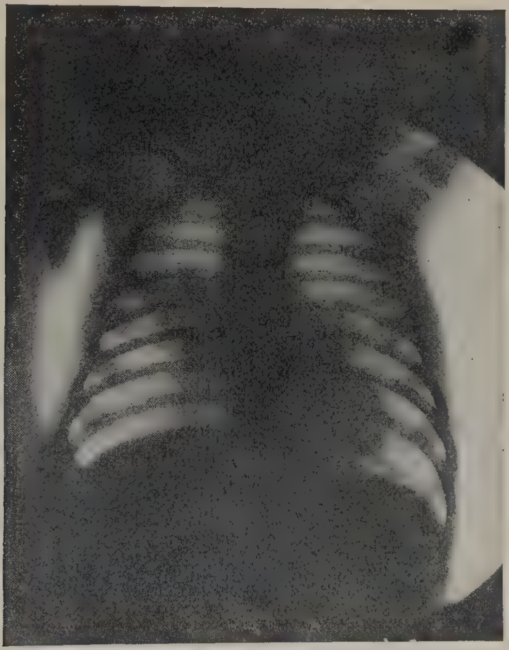

Fig. 81.—Atelectasis. Note dense shadows at the hilus both right and left—shadows are not characteristic of any one condition.

History: H. T.—five months of age—presenting symptoms cough—which had persisted for months—no history of acute respiratory infection.

Physical findings: Fair nourishment, no fever, dullness and bronchial breathing—normal blood picture—negative von Pirquet. Persistence of the symptoms and shadow over a period of five months. Diagnosis atelectasis by exculsion of other causes of consolidation.

Differentiation may be made from pneumonia by the acute course, with small amount of expectoration in the former, by a course of great chronicity in bronchiectasis, with abundant putrid expectoration, more marked in the morning. The disease of this grade and length secondarily produces congestion in the lung with involvement of the heart, hypertrophy and dilatation especially of the right heart.

Lung Atelectasis.—Atelectasis results from collapsed lung with little or no air content. Such areas naturally will produce shadows. Doubtless atelectatic areas occur in bronchopneumonia and may not be differentiated from areas of consolidation. But in the newborn, atelectasis gives a frequent and characteristic finding. A part or whole lobe may be involved with a rather dense shadow, more often involving the right upper lobe. Physical signs may be definite or they may be confusing. An airless area of lung overlying a large bronchus and acting as a good conducting medium may give dullness and bronchial breathing. On the other hand, a limited area not in contact with a bronchus may give diminished resonance and diminished or absent breath sounds. Fine crepitant râles are usually heard in both instances. There are no signs of inflammation such as enlarged regional glands. Adhesions and displacements of structures fail (Fig. 81).

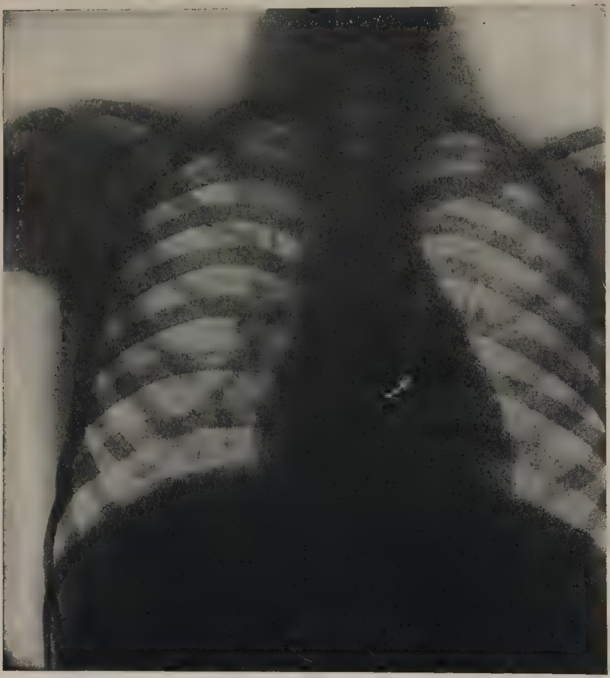

Fig. 82.—Foreign body (cartridge) in the left bronchus.

History: H. M.—age four years—two weeks before patient came to mother stating he had swallowed a cartridge—had a violent cough for the succeeding 24 hours. On entering the hospital there was little cough, "wheezy" breathing, normal temperature and patient was comfortable. Physical signs negative except for questionable decrease of breath sounds over the left lung. Following an unsuccessful attempt to remove the cartridge patient developed pneumonia and died.

Emphysema.—Emphysema in work with children is found accompanying pertussis, asthma, and as a vicarious process on the part of a functioning lung to compensate for other areas which are temporarily or permanently rendered airless. The symptom complex of old age—

emphysema with calcification of costal cartilages and subsequent inelasticity of the chest wall—we do not find in children. Plate or screen shows a low-lying flattened diaphragm, the costo-phrenic angle obtuse, or at least less acute than normal, and lung fields especially well lighted (see Fig. 85).

Foreign Bodies in the Respiratory Tract.—Foreign bodies in the trachea or bronchus. A very considerable literature has accumulated in the past few years pertaining to foreign bodies in the respiratory tract. The finding is often accidental, occurring during the routine examination for a chronic cough. At times the cough may be asthmatic in type. Again, the cough is productive of abundant fetid material suggestive of a lung abscess or bronchiectasis. The condition may be chronic for months or years, or very acute. The history may be clear as to the swallowing of an object or may not give a single clue. Such objects may cause a local infection and eventually produce an acute bronchopneumonia. The list of foreign bodies includes pins, needles, safety pins, buttons, coins, and so on. All cases of chronic cough or acute respiratory disturbances with an atypical history

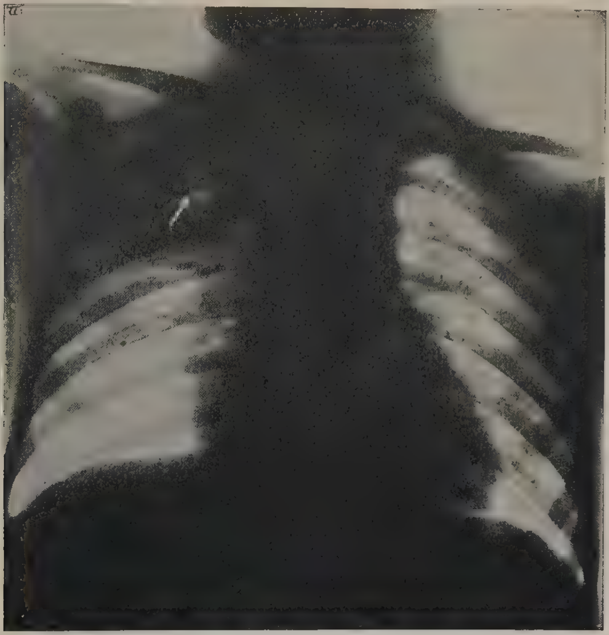

Fig. 83.—Lung abscess. An extensive shadow in the right upper lobe with indistinct margin fading out into normal lung tissue—in the center of the shadow a round well lighted area (1).

History: M. R.—eight years of age—three weeks previously tonsillectomy followed by irregular temperature malaise and persistent cough—one week previously there had been abundant, putrid, expectoration (rupture of abscess into a bronchus).

should be considered as demanding screen or plate examination. Shadows may be found in either main bronchus, but more often in the right (Fig. 82).

Lung Abscess.—For the diagnosis of this condition the x-ray is almost indispensable. Both screen and plate are helpful. With the screen one finds a rather circumscribed shadow, gradually fading out into normal lung structure. If the abscess has ruptured into a nearby bronchus, which nearly always occurs, in the center of the dark area a well lighted cavity may be made out. A dense shadow with horizontal level may show a wave-like motion on breathing or movement. This will clearly indicate fluid. If the abscess is to be treated surgically, screening or plates taken at various angles may afford a good degree of localization. From the author's experience lung abscess is rare in childhood and usually results from aspiration of material following operation about the mouth and throat such as tonsillectomy (Fig. 83).

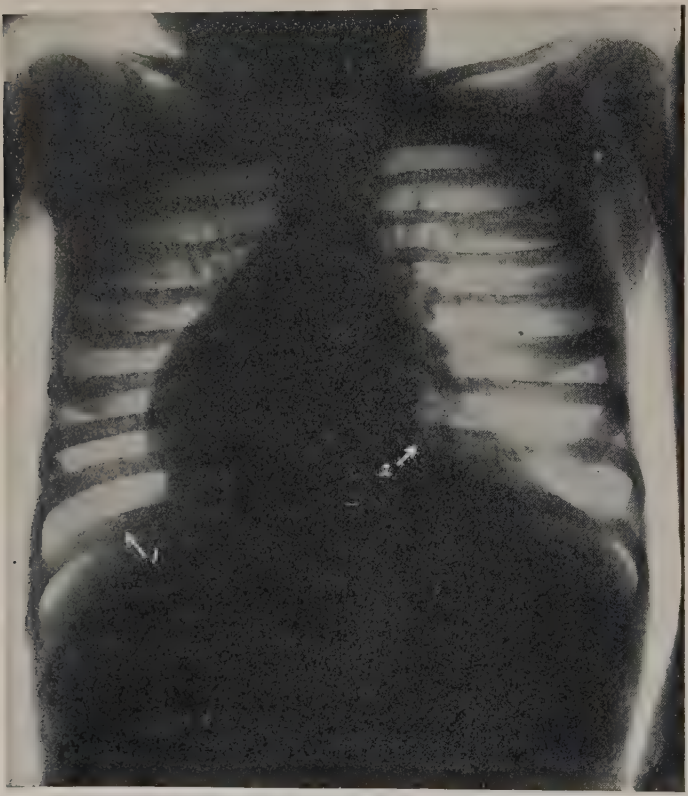

FIG. 84.—Diaphragmatic pleurisy. Note the normal curve of the left diaphragm (1)—on the right the diaphragm is hooked up to the heart with obliteration of the cardio-hepatic angle (2).

A phthisis cavity may be differentiated from lung abscess by a more sharply cut limiting wall. History and course of the disease are also of value. Evidence of old abscess may be a small radiating shadow with cirrhosis of the lung and little effect on pleural surfaces. An old healed tuberculosis cavity is shown by definite shrinking and retraction in the whole lung on the side involved.

Gangrene may occur following embolism and hemorrhagic infarction. Such lung areas of necessity produce shadows which, however, are not characteristic as to markings. Their general shape, location at the periphery, with involvement of the overlying pleura and the history, suggest the proper interpretation of the shadow, rather than peculiarities of the shadow *per se.*

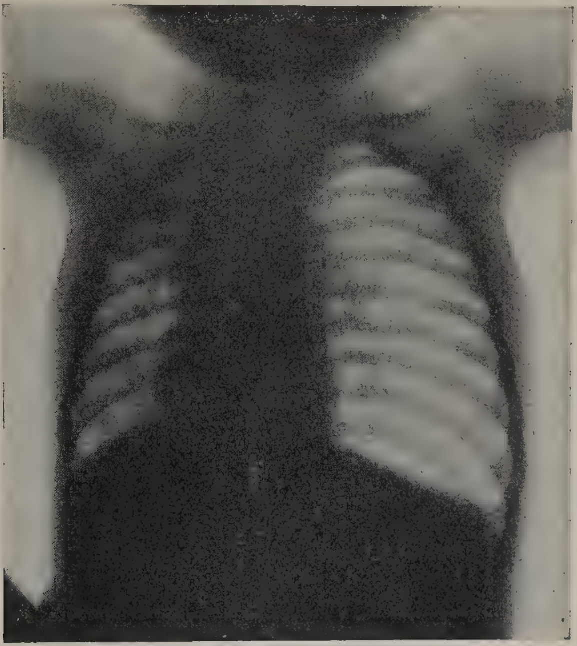

Fig. 85.—Fibrinous pleurisy with retraction. Plate B. M.—A rather diffuse shadow over the left chest (thickened pleura) while the right field shows compensatory emphysema—note the very faint shadows of lung structure and the flat diaphragm.

History of long standing tuberculosis—von Pirquet positive.

Diseases of the Pleura.—Acute fibrinous pleurisy gives no characteristic findings in the plate. The screen may, however, give definite evidence of trouble. Limitation of motion of the diaphragm and lesser lighting of the lung fields may be elicited when no pathological changes capable of producing shadows are found. Chronic fibrinous pleurisy in addition to the above findings, especially when involving the diaphragmatic pleura, may show adhesions between the diaphragm and pericardium. Such adhesions cause limitations of motion of the diaphragm on inspiration (Fig. 84).

An old fibrinous pleurisy with consequent thickening and adhesions of the pleural layers may cause confusion. At first sight the suggestion of lung consolidation is strong. The fluoroscope will show limitation of motion as in acute pleurisy but on deep inspiration one can detect some lighting up of the lung fields. Such pathology also leads to adhesion and consequent retraction with displacement of the heart and mediastinal structures. The displacement of the mediastinum is often plainly shown by a deflection of the band-like outline of the trachea (Fig. 85).

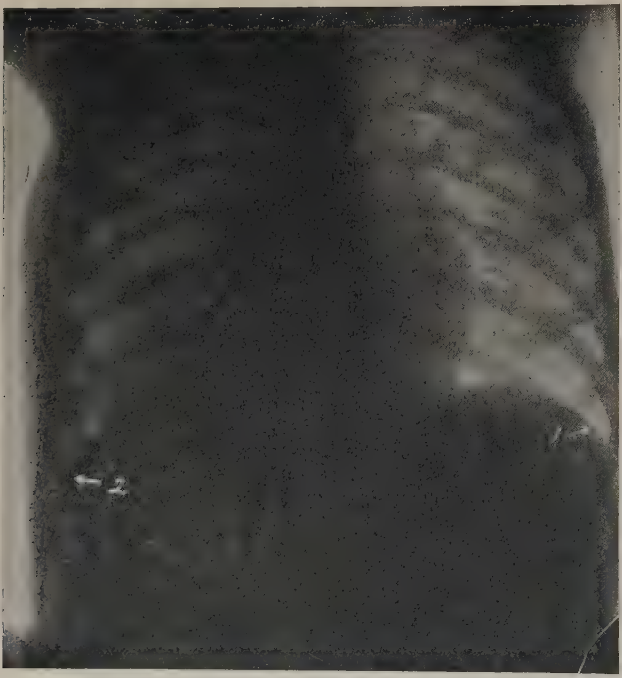

Fig. 86.—Large pleuritic effusion—empyema. Note the very dense homogeneous shadow in the entire left chest with marked displacement of the heart shadow to the right. Compare the normal costo-phrenic angle at the right (1) with its obliteration on the left (2).

History and course: C. B.—11 years of age—influenza in Oct.—entered hospital Dec. 9, with complaint of "pain in the stomach"—temp. 100^4—cough.

Examination: Dullness, diminished breath sounds and lessened fremitus over left chest—thoracentesis gave pus—recovery after rib resection and drainage.

Pleurisy with Effusion.—The demonstration of small pleuritic exudates is extremely difficult in children. It requires a quantity of about 100 c.c. or more before physical signs are present. However, the smallest effusions are made visible by the screen. This may require illumination at different levels, with the tube at different heights, since as we know, the fluid tends to collect posteriorly (see Fig. 43). The findings of a small effusion are: a dense shadow, triangular in shape over the base, not varying in its density with respiratory movements. The costo-phrenic angle, which is usually well lighted, even in consolidation, is obliterated by an effusion. Further, the screen will show diminished movement of the diaphragm on the same side. A rather large effusion gives a high, dense shadow, with sharply outlined upper limit, convex or S shaped, and narrower above, that is, wedge-shaped. The diaphragm stands lower and is much limited in its motion. A very large effusion produces a dense shadow which may completely obliterate the lung field, and might easily be confused with a massive pneumonia. However, effusion of such degree produces displacement of heart, mediastinum and trachea, away from the side involved. This displacement may be moderate or extreme depending directly upon the size of the exudate. Physical findings are often confusing also. In the infant a purulent collection in the pleural cavity produces bronchial breathing rather than diminished or absent breath sounds. Because

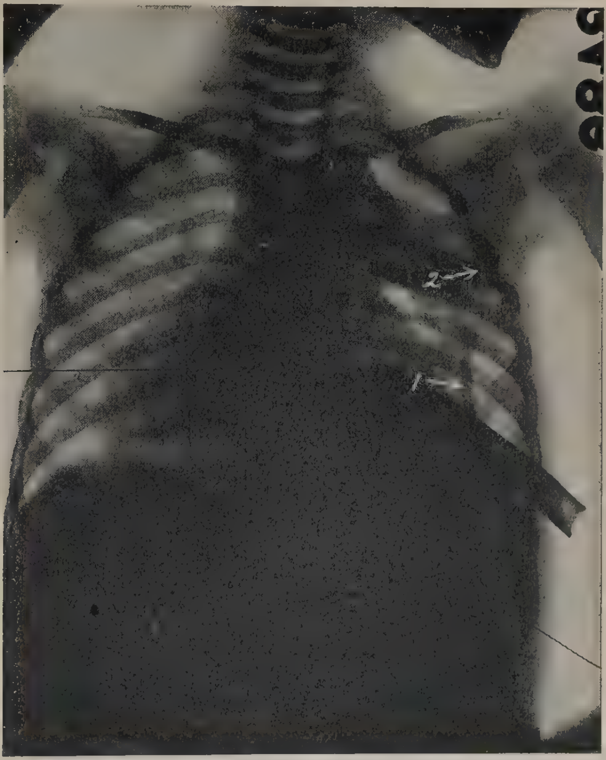

Fig. 87.—Pneumothorax. Note the well lighted space at the periphery and the sharp margin of collapsed lung (1). Pneumothorax was induced by rib resection and drainage—tube in place—lung held in contact with chest wall by adhesions (2).

of the resiliency of the chest, hard and careless percussion may fail to give the impression of fluid. The *x*-ray examination is of extreme value in this condition (Fig. 86).

From *x*-ray findings it is quite impossible to determine whether the fluid is an exudate, transudate or purulent collection. History, laboratory findings and thoracentesis must be called upon to differentiate between these conditions. Again, this illustrates the limitations of the *x*-ray. The roentgenologist may state the findings may show a shadow of a pleuritic exudate. Further he is not justified in going. The clinician must in the end interpret the shadow on the basis of other facts.

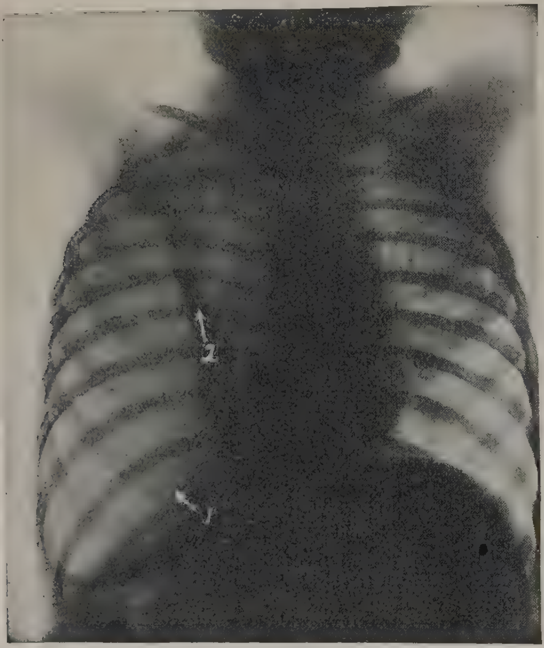

Fig. 88.—Pyopneumothorax. Plate taken with patient in prone position. Not the homogenous shadow in the left lung field without any natural lung markings. (1) The linear shadow (2) is the edge of the compressed lung.

Pneumothorax.—This gives a rather striking picture. On the side involved both screen and plate show a strikingly clear field. Closer inspection may show the lung collapsed and compressed toward the spine, while throughout the lung field the fine radiating shadows of lung structure entirely fail. With the screen there is found no change in degree in lighting up of the involved field on deep inspiration (Fig. 87).

Hydropneumothorax or Pyopneumothorax.—In this condition the screen examination surpasses the plate and gives almost dramatic findings. In the prone position, with dorsoventral lighting, confusion

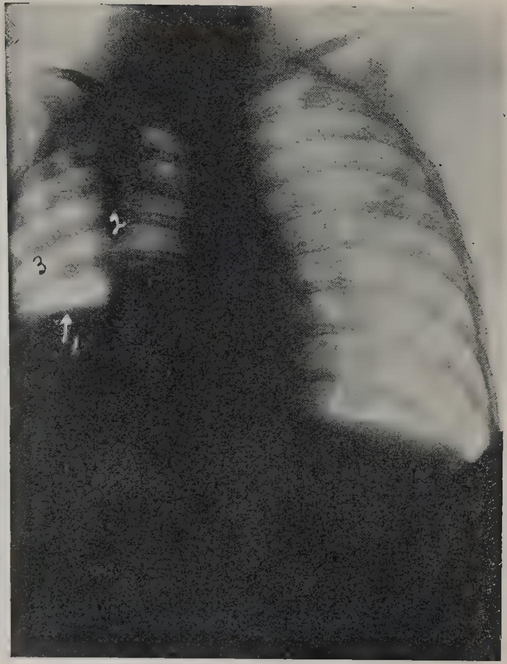

Fig. 89.—Pyopneumothorax. Same patient as Fig. 88—plate taken with patient in the upright position.

(1) Shows the horizontal line of fluid—(2) the compressed lung—(3) the air space.

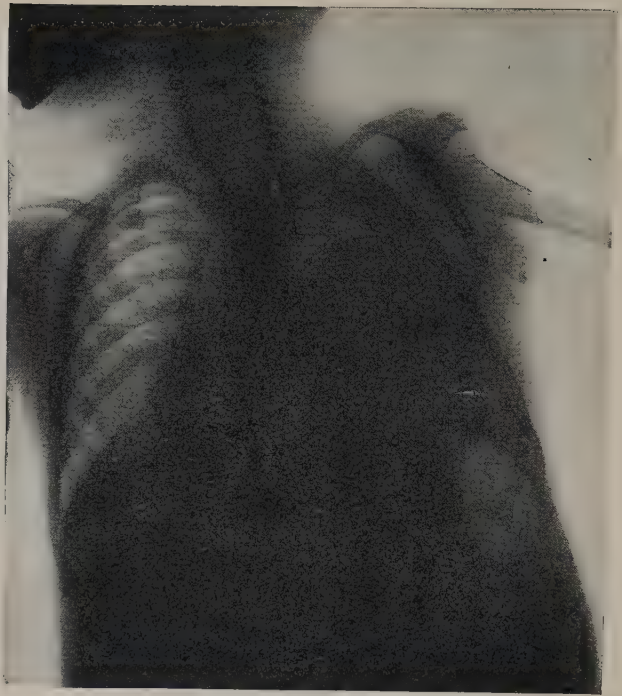

Fig. 90.—Empyema.

may arise. A rather dense shadow found throughout the lung field suggests a large pleuritic effusion or a massive pneumonia (Fig. 88). But in the sitting or standing posture the picture is characteristic. The lower chest shows a dense shadow, with sharply outlined horizontal border, and obliteration of the costo-phrenic angle, as in pleurisy with effusion. Above this shadow the field is unduly clearly lighted, while attention directed to the spine will demonstrate the shadow of a collapsed lung lying in contact with the spine (Fig. 89). Shaking of the patient will produce waves on the surface of the horizontal shadow. The fluid changes directly with changes in the position of the child. If a little patient is stood on his head the fluid flows to the apex, the air or gas collecting in the region of the diaphragm, the fluid being in fact free. A plate confirms the finding of the screen but shows no postural changes As with pleural effusions, the character

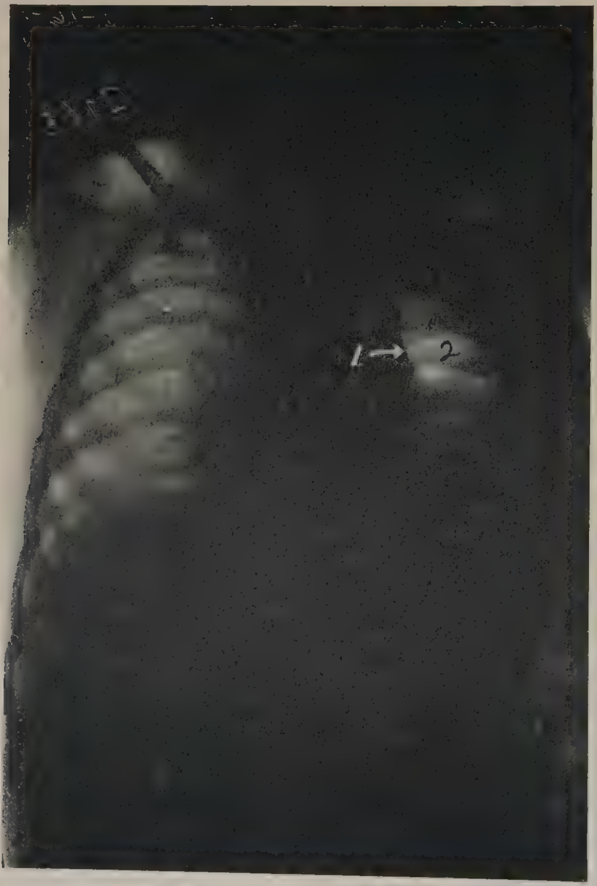

FIG. 91.—Same after paracentesis. Note the edge of collapsed lung (1) and the air space (2).

of the fluid must be determined by other methods. A pyopneumothorax is of more frequent occurrence than is supposed. Thoracentesis in children often results in the conversion of an empyema into a pyopneumothorax (Figs. 90, 91).

Interlobar Pleurisy with or without Effusion.—The diagnosis of this condition is made with certainty only by the *x*-ray. The finding is a roughly linear or band-like shadow extending from hilus to periphery and conforming to the topography of a lobe (Fig. 92).

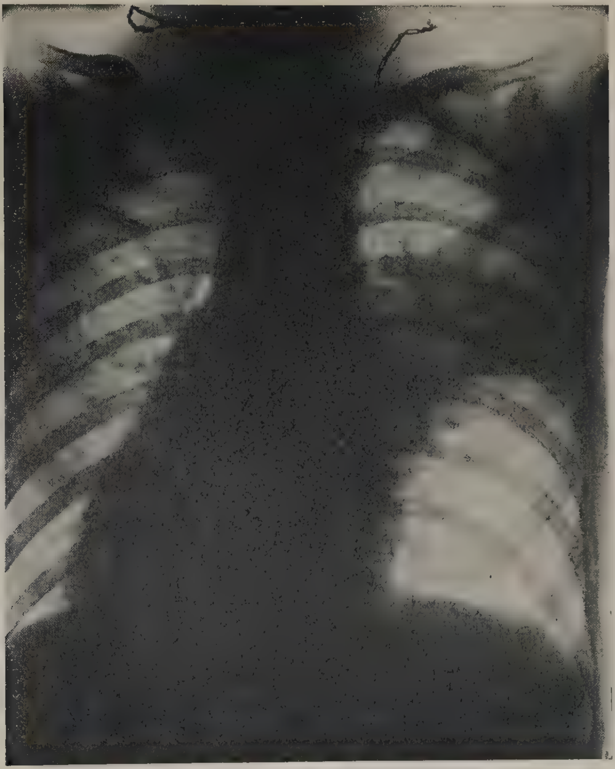

FIG. 92.—Interlobar pleurisy. Note the rather thin shadow in the upper right chest with sharp straight margin extending from root of lung to periphery at the anatomical location of the fissure between the upper and middle lobes. By fluoroscopy with the tube elevated and the screen lowered this shadow appears very narrow, almost linear.

History: M. P.—fever, cough, pain and practically negative physical finding. Uneventful recovery.

In the writer's experience all cases have appeared on the right side between the upper and middle lobes. Since the plane of the interlobar space is from above downward, the effect of transillumination varies with the position of the tube and screen. At certain levels the shadow is broad while if the tube is elevated and the screen lowered so that the rays parallel the plane between the lobes, the shadow may appear almost linear. If there has been a considerable exudate then a rather thick shadow may be found with convex borders. The use of the screen is of extremely great value in thoracentesis for pleuritic exudate of small amounts, and is almost a necessity in interlobar effusions. It can be of aid in larger effusions, not as to localization but in determining the amount of fluid. It is extremely hard to tell by ordinary clinical methods the size of the exudate. Further, if one does not take into consideration the differences in anatomy between the infant and adult, or control thoracentesis by fluoroscope, the diaphragm may be punctured. On the right side especially the diaphragm stands much higher than in the adult. The writer has seen such a mishap, the needle going through the diaphragm into the liver, as shown at autopsy.

Diaphragm.—A knowledge of the anatomy and physiology of the diaphragm is essential for interpretation of the *x*-ray findings. Its relative high position in the infant must be kept in mind. This may be still more exaggerated by enlargement of the liver and the presence of the "air bubble" in the stomach. The latter may push the left half of the diaphragm up an intercostal space or two. Massive pleuritic effusions may displace the muscle downward. Diaphragmatic pleurisy has been discussed. One may find occasionally a paralysis of the diaphragm from various causes such as injury or polio-encephalitis. Clinically the condition may be suspected from a limitation of the chest excursion and especially by a persistent ineffective cough coupled with a history of poliomyelitis, etc. Fluoroscopy will show a very high standing position of the diaphragm which moves but little or not at all on inspiration. If the paralysis is left sided, the heart will show displacement in its long axis. One or both sides may be involved.

Diaphragmatic Hernia and Eventration.—Pathologically these conditions are of three types:

1. True hernia, where the abdominal contents project through the diaphragm and are covered with a layer of peritoneum and pleura.
2. Spurious hernia, in which the serous coverings on the hernial structure are lacking.
3. Eventration, in which atrophic or defective development changes permit the displacement upward of abdominal contents (see Paralysis of Diaphragm).

Roentgenologically the condition is not so easy to recognize as one would suppose, and a differentiation of types of defect is barely a possibility. The fluoroscope may show a decrease of the usual respiratory movements of the chest and diaphragm, with mediastinal displacement toward the sound side. There may also be unusual shadows projecting into the lung fields. Solid contents of the viscera may give shadows, or air contents may produce unusual lighting effects. But these may fail. Introduction of a stomach tube or the administering of contrast material in the form of a barium mixture may permit one to make a definite diagnosis of the presence of particular abdominal viscera in the chest cavity.

Mediastinum.—Roentgen studies of the mediastinum are of extreme importance because of the great number of structures involved and their position of inaccessibility to the usual methods of physical examination. Anatomically it is the space lying between the lungs laterally, and the sternum and spine sagittally. The organs lying in the space with which we are concerned in our study are: heart, thymus, great vessels, trachea, esophagus, bronchial glands. Because of their relationship certain diseased conditions of the spine must be considered.

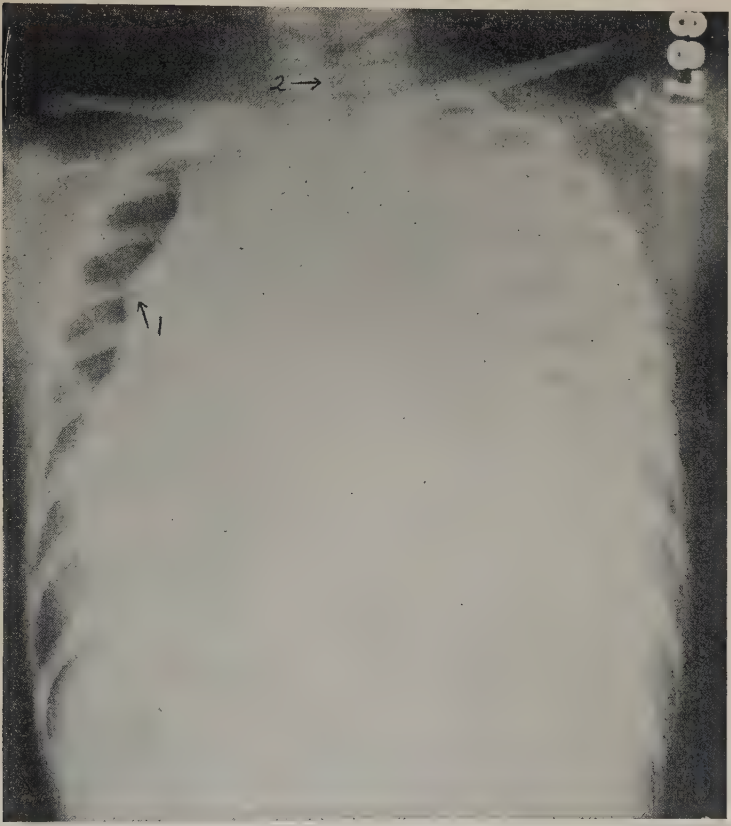

FIG. 93.—Leukemic infiltration of mediastinum. Dense shadow curving the center of the chest fading out on the right but with sharp curved margin at the left (1). Note there is no displacement of the mediastinum since the trachea takes a direct downward course (2).

History: M. L.—age seven years—presenting symptoms—swelling of face, extreme dyspnea, persisting for six weeks—appetite good—normal temperature.

Physical signs: Dullness over chest—more on right than on left—Hg. 80 per cent. White count 14,800—patient was given *x*-ray treatment. Courtesy O. W. Rowe, Duluth, Minn.

Tumors of the Mediastinum.—Inflammatory conditions of the bronchial glands have been discussed under the section dealing with tuberculosis. Tumors of the mediastinum which are nearly always primary in the glands or connective tissues are rare in infancy and childhood, although sarcoma, tuberculoma and teratomata have been observed in childhood. For a discussion of these conditions reference should be made to the roentgenological studies of adults. One condition which the pediatrist may anticipate is that of a leukemic infiltration of the mediastinal glands. Shadows in such cases are very dense, homogeneous, with sharp margins and often showing rounded curves rather than irregular outlines. The trachea is not usually displaced laterally. As proof of the leukemic nature of the neoplasm the changes in the shadow under treatment with x-ray may be noted (Figs. 93, 94).

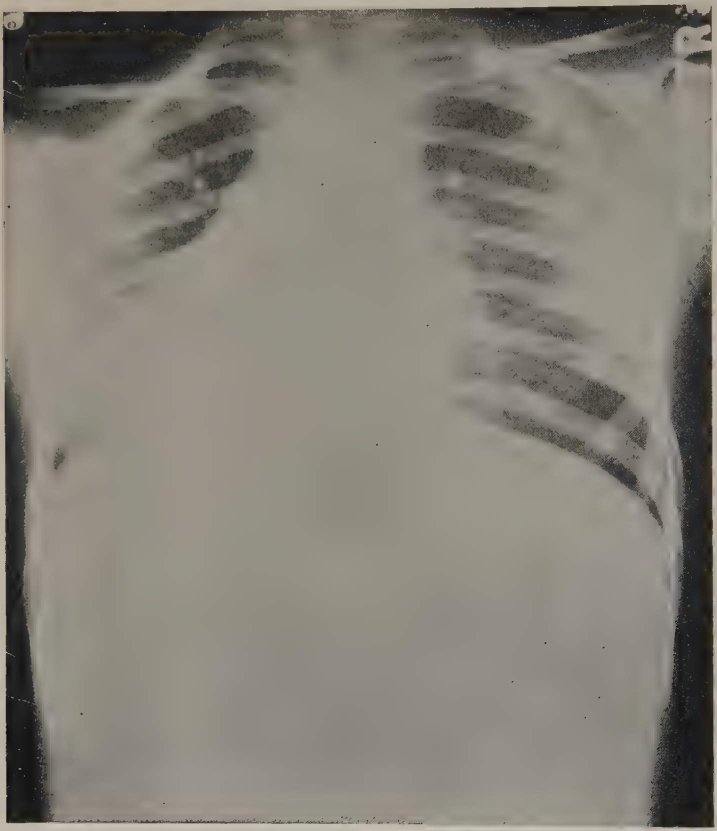

FIG. 94.—Case 43 after x-ray therapy—this plate taken two weeks after that illustrating Fig. 85.

Note almost complete disappearance of the shadow except at the base. At this date the white count was 99,000 of which 98 per cent. were lymphocytes, confirming the diagnosis of lymphatic leukemia. Exitus one month later. Courtesy Dr. O. W. Rowe, Duluth, Minnesota.

Fluoroscopic examination in the right anterior oblique position may permit of more or less differentiation of the mass from other structures.

Thymus.—The thymus grows from birth until the age of two years, after which it decreases in size and undergoes degenerative changes. In adult life it is a rudimentary structure not visible with *x*-ray unless it becomes the site of neoplastic changes. In the infant the gland is capable of producing a shadow, especially if it shows hyperplasia. To demonstrate the thymus with the plate requires especial technique, such as instantaneous exposure and careful development, for at best its margins may be indistinct. The thymus lies back of the sternum at its base and extends down over the heart, the right lobe being more prominent than the left. It may be revealed both by plate and screen. When is the shadow pathologic? What degree of enlargement is necessary to produce symptoms? This is a difficult problem in roentgenology. The writer has diagnosed enlarged thymus to find at postmortem the gland to be normal in appearance and of average weight according to figures prepared by Gundobin. Further confusion arises in the roentgenogram if there

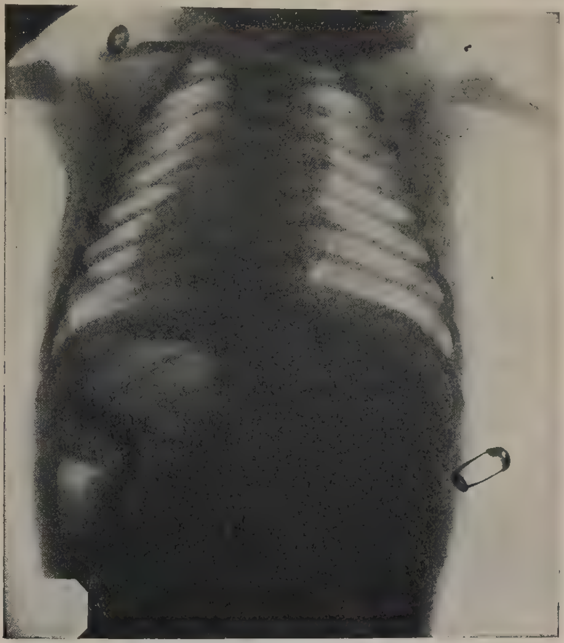

Fig. 95.—Over the base of the heart an indistinct broad shadow extends up into the neck region—at the margin the shadow is fainter—the heart shadow appears normal as to size, shape and position.

History and physical findings: Baby J—newborn, shows cyanosis, dyspnea, dysphonia, retraction of lower chest on inspiration—dullness over the base of sternum in the thymus region. Symptoms gradually disappeared during following weeks.

has been the slightest rotation of the patient at the time of exposure. Even a slight rotation projects the shadows of the great vessels at the base of the heart to right or left, giving the appearance of an enlarged thymus shadow (Figs. 95, 96).

An enlarged thymus may be found in status lymphaticus, which, however, is a rather indefinite symptom complex. It may also produce inspiratory and expiratory dyspnea, the so-called thymic dyspnea. This is not distinctive from the respiratory interference caused by enlarged bronchial glands. Hochsinger in 1904 suggested that congenital stridor was also due to enlarged thymus, but later studies fail to confirm this conclusion (Fig. 97).

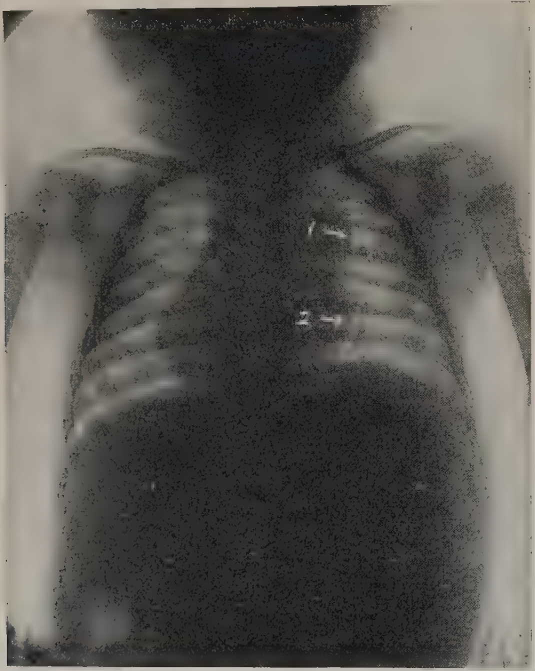

FIG. 96.—Thymus shadow. Note the broad shadow over base of the heart extending up into the neck region. Interpretation is rather difficult here because the plate shows rotation and consequently displacement of the heart and mediastinum to the right. However, it is to be noted that the shadow in question (1) projects to the right beyond the line of the right heart border (2) and is in all probability that of an enlarged thymus.

Diseases of the Esophagus.—There are four disorders of the esophagus which are frequently met with in pediatric practice in which the use of the *x*-ray may render signal service. Roentgenology has broadened our views on these diseases. These conditions are:

1. Foreign bodies in the esophagus.
2. Congenital atresia.
3. Acquired stenosis.
4. Cardiospasm.

Neoplasms, such as carcinoma, which are frequently found in the adult are rarely if ever found in childhood.

I. Foreign Bodies.—It is a matter of common knowledge that infants and children swallow a wide variety of objects, from pins to wrist watches. It is surprising what large objects may be swallowed without difficulty, while again small objects may be arrested in their descent from the impinging of sharp points, or an associated cardiospasm. Fluoroscope or plate will demonstrate the location and character of objects. There is no call for especial skill in interpretation. Symptoms of a foreign body in the esophagus may be remote from the time of swallowing the object. This is most apt to occur if the object is small enough to permit of the passage of food. In such an instance the object may later produce a spasm or inflammatory reaction with consequent scar retraction. This results in a local constriction, and above it a sacculation of the esophagus. Clinically this is shown by the ability to swallow only a small amount of solid food, which may later

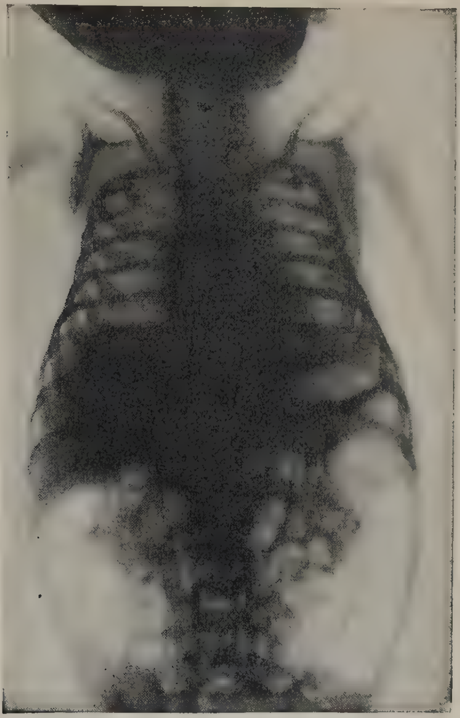

Fig. 97.—Congenital stridor. Note position and course of the rib shadows due to the sinking in of the manubrium sterni with inspiration—no thymus shadow visible.

History: A. H.—Male—11 days old—with inspiratory stridor. At one year of age the stridor was extreme. Patient died at the age of 13 months. Postmortem report "no anatomic evidence as to the exact cause of death." The thymus weighted 17 G. which is within the range of the normal weight.

be recovered by regurgitation. Liquids may pass the point of constriction while solids fail to do so. Inanition and loss of weight follow. Obscure persistent regurgitation, with loss of weight and lack of dyspeptic symptoms should call for an *x*-ray examination. Plate or screen will reveal the object, but not the condition of the esophagus unless contrast material is introduced into the esophagus. Older children will readily swallow a mixture of cereal and bismuth or milk and barium (see Diseases of the Stomach). In younger children the substance may be introduced by a stomach tube. With such preparation a plate or fluoroscopic examination will outline the esophagus. At the point of stricture the barium shadow fails, while above will be found the broadened or globular shadow of a sacculation. If the lumen is not entirely occluded the shadow of a thin stream of the contrast material may be followed past the constriction and into the stomach (Figs. 98, 99).

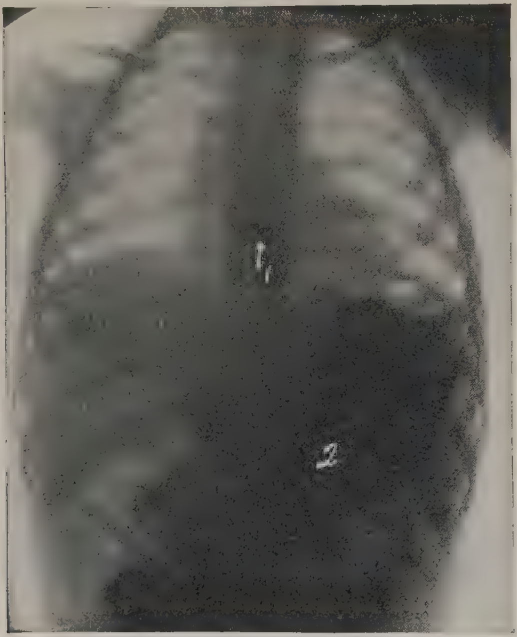

FIG. 98.—Foreign body in the esophagus. Shadow of foreign body in the esophagus (hook used for dress fastening). (1) Also barium shadow in the stomach. (2) Outlines of esophagus not visible.

History: R. H.—Male 14 months—well until two months ago when the child vomited mucus—most of food was retained—later regurgitated more food—lost weight and became very constipated. Patient was treated for dyspepsia for one month and the foreign body was discovered in a routine fluoroscopic examination—it was not suspected.

II. Congenital Atresia.—Clinically this may be suspected from the history of inability to swallow more than a few cubic centimeters of milk, regurgitation, loss of weight, and absence of fecal material after the passage of the meconium. Failure to pass a small tube into the stomach may give further proof. But the fluoroscope or roentgenogram of the chest after the giving of a small amount of barium and milk mixture by mouth or tube allows one to visualize the defect as to form and location. In contrast to acquired stenosis the congenital atresia will show a complete obstruction and no shadow of contrast material in the stomach (Figs. 100, 101).

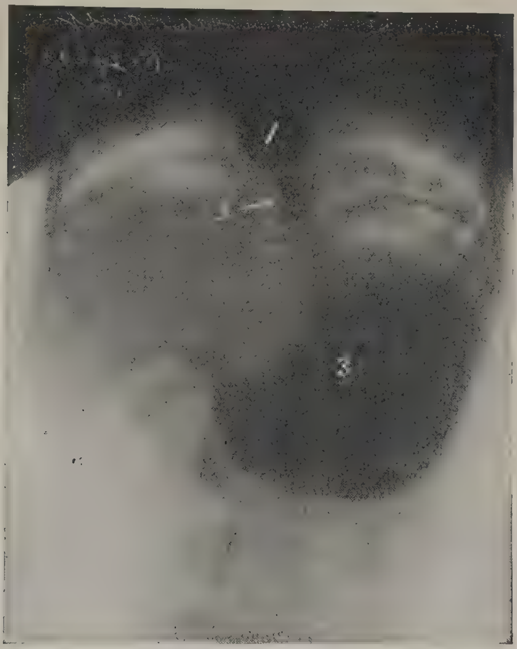

FIG. 99.—Same patient as Fig. 98 during the taking of a barium mixture. Note the dilated esophagus (1)—thin stream of barium mixture passing the point of construction (2) and a considerable of the contrast material in the stomach (3) after removal of the eye and dilatation by the use of bougies the patient made a complete recovery.

From postmortem studies it has been shown that the pathological anomaly concerns the trachea as well as the esophagus. The atresia occurs usually at the level of the point of bifurcation of the trachea (between the third and fourth thoracic vertebræ) and there exists in one form or another communication between the esophagus and the air passages, either trachea or large bronchi. This connection occurs through the lower segment so that no help can be expected from gastrostomy unless the esophagus is ligated at the cardia. Air may be swallowed and pass into the stomach or gastric secretions may be regurgitated in the air passages (Fig. 102).

III. Acquired Stenosis.—Acquired stenoses other than those resulting from lodgment of a foreign body occur following the swallowing of caustics of low toxicity, usually a solution of lye. The clinical history is usually clear. Following the ingestion of the caustic, the child suffers pain and has difficulty in swallowing food, from the accompanying swelling and pain. Subsequently the dysphagia progresses, especially for solid food. Eventually regurgitation, loss of weight, obstipation occur (as described under I). Older methods of diagnosis consisted in the passage of bougies. In the acute condition this is especially dangerous because of the erosion and the possibility of breaking through into the mediastinal space. All the information is obtainable with safety by means of the contrast material as described above. Older children may be fluoroscoped in the anterior oblique position while swallowing the mixture. The bolus may be followed down to the point of stricture. Observations may be made as to the degree of

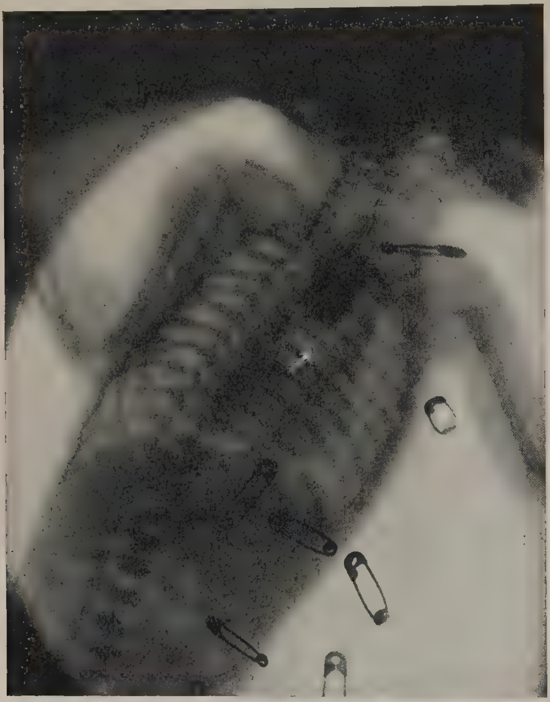

Fig. 100.—Congenital atresia of esophagus. Plate shows barium mixture in the esophagus—rounded margin shows complete occlusion. Note the line of the trachea over the mass and the bifurcation of the trachea just at the lower level of the shadow (1). The general relationships appear abnormal which is due to the fact that the plate was taken with the child held in the anterior oblique position. There is no sign of air in the stomach.

History: Newborn—presenting imperforate anus which was relieved by operation. (Atresia along the digestive tract is often multiple.)

The infant was unable to swallow. After passage of meconium there followed no fecal material—stomach tube could be passed only a few cm.—operation was refused—patient died on the seventh day.

constriction, noting the amount passing by into the stomach. A plate taken in the same position or by dorsoventral exposure confirms fluoroscopic findings and gives a record. Progress of the case may be checked by subsequent exposures (Fig. 103).

IV. Cardiospasm.—Cardiospasm is occasionally met with in children. This is evidenced by arrest of the bolus just before entering the stomach. Though there is a neurotic element in the etiology there

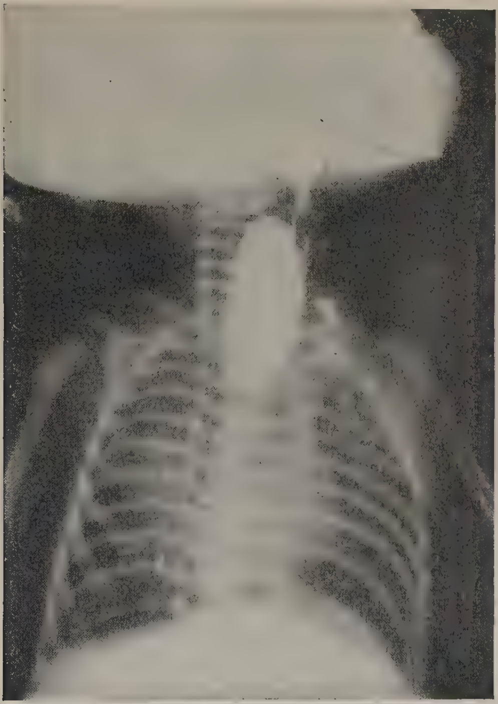

FIG. 101.—Congenital atresia. Baby H. Newborn—note the shadow of bismuth in the esophagus, complete occlusion as in Fig. 102. Courtesy of Dr. O. W. Rowe, of Duluth, Minnesota.

arises the possibility of there being primarily an injury or foreign body.

The *x*-ray findings are quite the same as those for acquired stenosis, but they are usually not constant, repeated examinations demonstrating remissions. This at once rules out the possibility of organic changes.

Prevertebral Abscess.—This condition is not frequently met. It may produce a wide variety of symptoms such as dysphagia, dyspnea, chronic cough, asthmatic attacks. Clinically it may be impossible of diagnosis without the *x*-ray, especially in the earlier stages. Pathologically it depends upon a cervical or thoracic Potts disease. Pus collects beneath the prevertebral fascia and bulges forward and burrows downward very like a psoas abscess. The abscess may precede any symptoms of breaking down of the vertebræ such as angular deformity and pain. With the fluoroscope in the anterior oblique position one sees the shadow of the spine with a mass bulging forward. Over the mass anteriorly the trachea may be traced. Such a finding serves to differentiate it from bronchial glands. The plate by dorsoventral exposure shows a more or less globular mass, unless obscured by the heart shadow. Later in the disease a plate will show bone changes (Fig. 104).

In several instances the writer has found such a shadow in the course of routine examination when such a thing was not suspected.

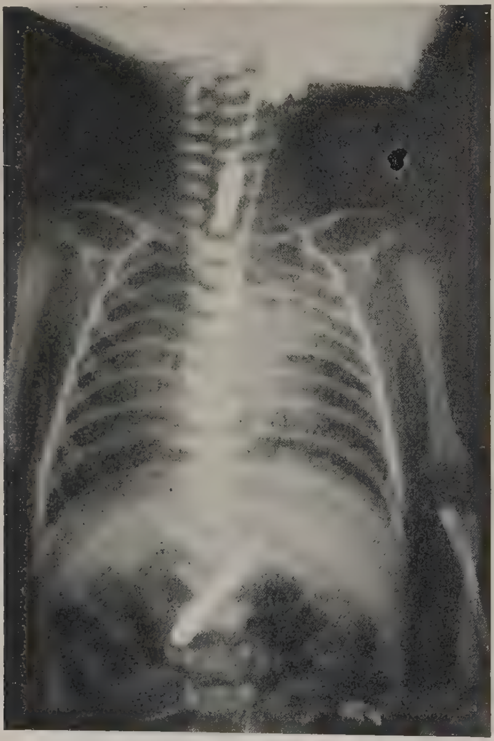

Fig. 102.—Same patient as Fig. 101. Postmortem, barium was injected into the esophagus through the stomach—the contrast material passed up the lower segment of the esophagus into the trachea and then into the lung. Note the fine barium shadows throughout the lung fields. This is the inosculating type of the disease. Courtesy of Dr. O.W. Rowe of Duluth, Minnesota.

This illustrates the value of a complete x-ray examination in obscure conditions producing cough, dyspnea, and dysphagia.

Funnel Breast.—Bony deformities of spine and chest will naturally cause displacement of the chest viscera with corresponding distortion of the normal roentgenogram. Much confusion of physical signs may result also as one is forced to admit after examining a case of scoliosis of severe degree. Such deformities include scoliosis, lordosis, kyphosis

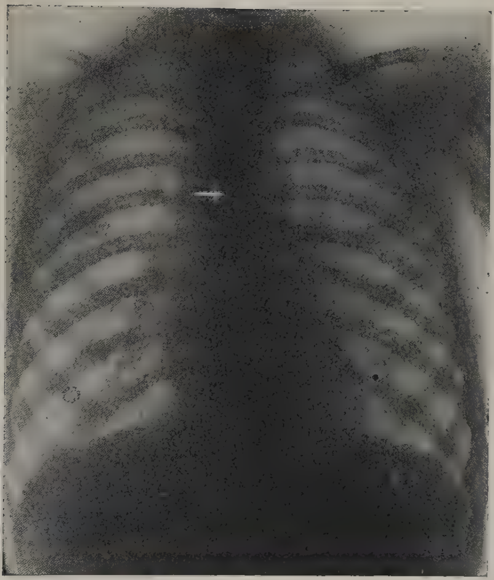

Fig. 103.—Acquired stenosis of esophagus from swallowing lye. Note shadow of dilated esophagus occlusion is not complete—there is a very narrow shadow of the barium mixture trickling by the stricture (1).

History: Male—age four years—two months ago swallowed lye—for two days took no food—then ate fairly well—lately could swallow only small amounts of liquid—regurgitated solid food—has emaciated rapidly.

A large bougie was easily passed after which the child was able to swallow readily. This indicated either a very mild stenosis or else an associated spasm of the esophagus.

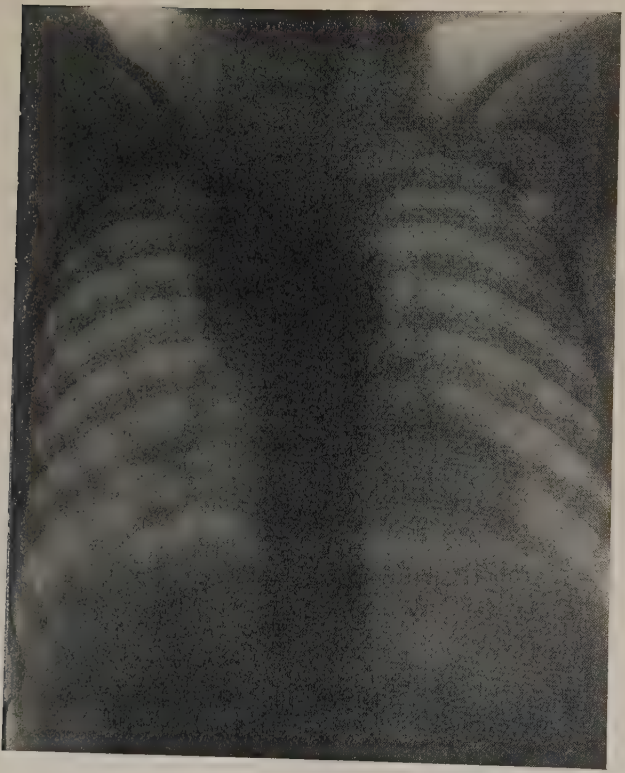

FIG. 104.—Prevertebral (thoracic) abscess. Lung fields and heart shadows normal—over the base of the heart in the mid-line a large, dense almost globular shadow, the outline of a collection of pus over the anterior surface of the spine.

History: Male child—eight years of age, entered the hospital with the complaint of asthma. Moderate cough—no complaint of pain—child played actively—physical findings were negative except for a positive D'Espine—reflexes were normal. The von Pirquet was positive and closer examination revealed slight limitation of motion of the upper thoracic spine.

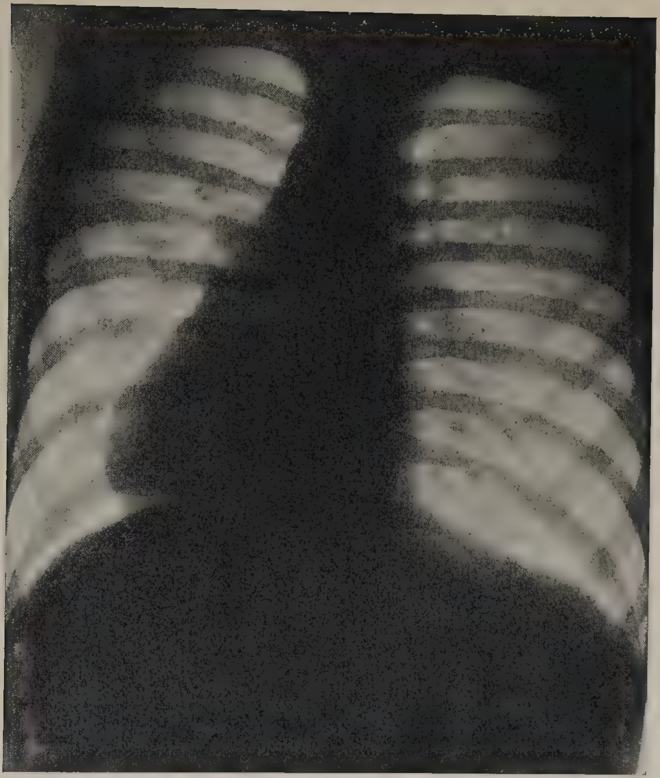

Fig. 105.—Note displacement of the heart to the left—the right border not visible since it lies within the shadow of the spine. The change is evidently not inflammatory since the lung fields are clear—there are no gland shadows nor evidences of adhesions.

History: D. B.—Male, eight years old—presenting symptoms "stomach trouble and flat chest"—physical findings, funnel breast of extreme degree—heart displaced to the left—inequality of the radial pulse—the right being weak and irregular.

and changes in the sternum such as pigeon and funnel breast. A plate will often serve to give one a true picture of the visceral relationships (Fig. 105).

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CHAPTER XVI

PECULIARITIES OF DISEASE IN CHILDHOOD

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Disease in infancy and childhood differs very much from disease in adult life. This is especially true under the eighth year, after which the differences gradually lessen, and by the twelfth or fifteenth year (puberty) disease coincides with that of the adult.

The peculiarities of early life are dependent upon heritage, birth, the undeveloped body and functions, the necessary orientation to the new outside surroundings, upon metabolism and nutrition, and upon both mental and physical growth and development.

The new-born infant is brought into the world in an undeveloped condition. It is suddenly changed from a temperature of 98 degrees Fahrenheit to one of 68 to 72 degrees. It emerges from a bland aqueous environment into one of dry air, and just as suddenly its food and oxygen come from a vastly different source. The parasitic means of protection and supply is changed to a self dependent one. The shock of this change falls first directly on the epithelium of the tender skin covering of the body surface, which in proportion to weight and size of the body is much greater than that of the adult. Consequently, there is greater heat dissipation, or more absorption of heat, depending upon the surrounding temperature. Secondly and thirdly the effects of this sudden change fall upon the delicate mucosa of the respiratory and digestive tracts, whose operations of functions too are abruptly established.

Indirectly the shock falls upon the internal organs of the body. These are protected from varying temperatures, mechanical and chemical irritants and infections by the proper functioning of the epithelial coverings.

So long, then, as the epithelium is able to cope with the changing temperature, the onslaught of irritants and bacteria, and so long as the general nutrition is good, the body will be healthy. If the epithelial coverings do not perform their expected functions, or the external conditions be overwhelming, disease will ensue. Thereupon, the internal or deeper organs, which do not possess the same power to react to variation of temperature, irritants and infections, become disordered and the body temperature, fluid and chemical constituents become abnormal.

Thus we see that the general body health is dependent upon the proper reaction of its different tissues, which in the infant are of the embryonal type, richly supplied with blood and with an imperfectly developed nervous system. Hence the frequency of hemorrhages in infants. The lessened receptiveness of the nervous system with decreased reflex action and scarcely any inhibitory function readily permits loss of nervous equilibrium. Therefore in infancy nervous instability is constant.

The general muscular action, both voluntary and involuntary, is less, and is imperfectly controlled. The infant heart, however, is proportionately larger and stronger, while the arteries are of greater caliber and lesser length; hence the blood circuit is shorter, and the absorption and reaction from it the more rapid.

Digestion in the infant is not so complete as in the adult because muscular action is defective and digestive secretion is less. It is dependent upon mother's milk, and its adaptability to any other is more or less difficult. Unfortunately artificial feeding is usual in a large proportion of infants. The foreign food, with the accompanying profuse intestinal bacterial flora, may quite readily derange the largely undeveloped intestine, breaking down its slight resistance and allowing infection to gain entrance. Thereupon not only the digestive tract is affected but also the entire body suffers from lowered vitality.

Proportionately, the lymphatic system of the infant is of greater extent than in the adult; both the nodes and lymph vessels are larger. The tissue is much more porous and loosely connected, with less power of reaction. Absorption, of course, is greater, and inflammation and overgrowths are common.

Thus we find the new-born baby undeveloped in tissue and in function, extremely susceptible to disease, and demanding special protective environment, hygiene and food. It is not a miniature edition of the adult—it differs anatomically and physiologically. The premature infant is naturally even more undeveloped than the full-term baby. It is out of its physiologic environment, consequently its power of resistance is so lowered that any slight disturbance might be fatal.

Heredity has exerted definite influences upon the offspring. It has transmitted through the generating cells the individual peculiarities of the progenitors as well as definite characteristics of the family, race, and species. It has pre-controlled, by the definite arrangement of the protoplasm in the germ cells, the fetus' development in visual likeness to the parent as well as peculiarities of anatomic structure or physiologic function. Because of these inherited peculiarities the predisposition toward certain disease is the same in the offspring as in the parent. Disease in itself is not inherited, nor is the predisposition to disease, but the structural peculiarities are innate and they possess tendencies to disease. For instance, excessive lymphatic tissue appears in families. It is transmitted from parent to offspring, predisposing to hypertrophied tonsils and adenoids, adenitis, and otitis media. Inherited structural defects in the retina cause color-blindness, and inherited defects in the structure of the blood and vessel walls give rise to hemophilia. The disposition to indigestion may be due to an afunctioning or to an absence of one or other of the digestive secretions, the products of an inherited cell defective either in structure or in nerve stimulus. In epilepsy or insanity the inheritance is not of the disease per se, but of faulty cerebral tissue or of other abnormal tissue limiting or preventing the brain development. The neuropathic child has inherited a hyperexcitable nervous structure.

Peculiarities which are inherited must have been congenital in the parent and grandparents. They cannot have been the peculiarities which the parent acquired. For instance, a parent may have had upon birth an amputated foot which was acquired in utero, or the parent may have had scarlet fever and therefore acquired an immunity to that disease, but these conditions are not inherited by the offspring. Disease may be transmitted from parent to child, but as such it can never be inherited. Thus syphilis is transmitted in utero from mother to fetus, and tuberculosis is a post-natal infection. They are not inherited.

Inborn diseases are those conditions which befall the fetus in utero. Most important of them are the defects in development and the malformations. The former impair very much the viability of the child, while the latter are more or less detrimental to locomotion. Why there should be defects in development is imperfectly understood, but there seems some good reason to mention degeneracy and pathologic processes, such as rickets, in the parents. Most common of the developmental failures are hare lip and cleft palate. Others are meningocele, spina bifida, congenital heart conditions, hypospadias, epispadias, extrophy of the bladder, and atresia of the rectum.

Malformations such as club foot or hand, and congenital dislocation of the hips, are due to cramped positions, as in narrow uteri, which may be inherited. Therefore malformations may appear in families. Pressure from the umbilical cord accidentally wrapped around a part of the fetus may cause paralysis or amputation of that part.

In passing through the birth canal the child is subject to injuries from pressure or from necessary manipulation in delivery. There may be pressure or hemorrhage causing paralyses, dislocations and fractures of the bones, and lacerations of the skin.

The newly born may suffer asphyxia either from some obstruction, as by mucus in the upper respiratory tract, or from lack of stimulus to the respiratory center in the brain. Asphyxia may occur in utero, as in cases of placenta praevia. Atelectatic areas occur frequently. Infection of the umbilicus may quite easily occur, from which general sepsis might follow. The respiratory and digestive tracts may become infected from inspired or swallowed mucus. The eyes are prone to infection should gonorrhea be present in the mother. Jaundice is constant, to a moderate degree, in the newly born, but if persistent and increasing there is probably some catarrhal blocking of the bile ducts, or a malfunction of the blood or the liver.

Among the most important factors of etiology in disease of infancy are improper feeding, unhygienic surroundings, and neglect, from which gastro-intestinal, respiratory, and nervous disturbances arise. These may develop rickets, scurvy, or marasmus, and predispose to recurrent acute disorders.

Growth is the most characteristic feature of the young and general changes are greater in the infant; therefore the food must be sufficient and competent, the surroundings clean and wholesome, and the attention efficient and intelligent.

Infection is a conspicuous cause of disease in early life, because of the imperfect development of the various organs, and because there has been no immunity established by previous attacks. Infancy, childhood, and youth are more susceptible to all infections, and for this reason the acute infectious diseases predominate at this time. Exposure is as much or possibly greater than in adults, while fear and precaution are unknown to the child.

The age of puberty is attended with many dangers to health. The changes in the organs are sudden. The heart grows larger, the blood-vessels become narrower, and the blood pressure increases. The head and body grow and the muscles gain strength. The sex organs strengthen and new impulses arise. Metabolism is very active and at its height. At this time particularly mental disorders may develop and hereditary defects appear. Anemic conditions arise and may be followed by constitutional diseases. It is a most strenuous period both physically and mentally.

There are few acute pathologic lesions found in children that are not, at least now and then, seen in adults. Chronic changes of heart, kidneys, and liver so frequently found later, are never seen. Arteriosclerosis is unknown and tumor growths are very rare—especially carcinoma, which is almost never seen. Sarcoma is not infrequent. There is, however, a constant and peculiar distribution of disease involvement to certain tissues. The mucous membranes are affected frequently, the serous membranes not so frequently, except that empyema, peritonitis, and otitis media are not uncommon disorders in children. The gastro-intestinal mucosa suffers much more frequently in summer, while the respiratory tract lining is most commonly involved in winter. Then too, in children any debilitating disease of one or two weeks duration is always complicated by some pneumonitis.

The skin is frequently involved with hemorrhages, infiltrations, and pustulations. Irritations, exanthemata, eczema, and parasitic infections are common in children.

The lymphatics, in acute infections, are very readily inflamed, and frequently suppurate. The nodes (cervical, tracheo-bronchial, and mesenteric) are common sites of acute tuberculous infections, showing marked enlargement, caseation, and breaking down. In chronic disease, as syphilis and tuberculosis, the lymph nodes are constantly hypertrophied and fibrous.

Tuberculosis has a predilection for the growing bones as well as for the lymph nodes. The involvements are peculiar to childhood. The bodies of the vertebræ, the hip, the ankle and knee-joints are the most usual locations affected—beginning as a tubercle in the marrow of the epiphyses and spreading at the periphery by daughter tubercle formation, the area envolved, breaks down at the center, and becomes necrotic, and unless the process is arrested there follows a destruction of the head of the bone.

Syphilis of the bone is common in children. Most usually it is seen as an osteoperiostitis, a diaphyseal involvement instead of a joint involvement. But very early in congenital syphilis, however, one often finds synovial bone destruction, while later there is the periosteal infiltration and bony thickening of the shaft, which may break down late and form definite sinuses. The diaphyseal involvement is seen most frequently.

An acute epiphysitis is common in children. It is traumatic, non-bacterial or bacterial secondarily. The growth line of the bone is easily injured by excessive exercise, and infection may enter, proceeding rapidly to suppuration.

Scurvy seen in infancy involves the periosteum, with hemorrhagic infiltration. It may be an epiphysitis, suppurative or non-suppurative. Rachitic bones are soft and show an unnatural flexibility. The epiphyses are enlarged, especially the lower end of the radius and tibia, and at the costochondral junction. The long bone shafts are thickened and bowed. The frontal bones show embossing. The epiphyseal parietal line of the long bones is destroyed and cartilage remains instead of calcified bone having been formed. Fibrous and osteoid tissue is produced.

The acute inflammatory infections of the meninges are more common in early than in adult life. Tuberculous and pneumococcic meningitis are much more frequent. Polioencephalitis and poliomyelitis are almost peculiar to childhood, probably because the adult has become immune. Hydrocephalus, both internus and externus, originates in infancy. Hemiplegia and chronic degenerative changes are infrequently seen, although hemiplegia in childhood does occur in syphilis and pertussis.

Up to the fourth year the heart rarely shows any pathologic lesions other than congenital developmental failures. In later childhood the acute processes (endo, myo, and pericardial) seen in adults are encountered quite frequently, but never the chronic ones. The mitral valve is most frequently involved.

All symptoms of disease in children are more severe and intense than those of disease in adults. Although the cause of the disease be the same the effects vary in symptoms, intensity, and course. The young tissues are undeveloped and in addition to wear and tear are most actively concerned in growth and development. The nervous control is imperfect. The excito centers are acutely active and unrestrained by inhibito reflexes. Immunity, which the adult has acquired, is absent in the child, so that disease has an innocent, unguarded, and uninvaded field.

As a rule disorder in a child has a more violent onset, with a higher fever than in an adult. Vomiting occurs with great frequency and upon the slightest provocation. Cerebral excitement or severe depression appear often and convulsions or meningismus are quite readily provoked.

Ailments in early life usually are simple and uncomplicated so that as a rule one diagnosis will cover the condition. There is not so much cardiac debility and renal impairment, and arterio-sclerosis has not occurred as in adults. There are not the physical and mental abuses to which the adult subjects himself. The child is natural and truthful, unprejudiced, and seldom influenced by mental impressions and emotions. The adult is often prejudiced and misleading, though perhaps not intentionally so. The child is not a mere immature adult. Its nature and ailments are simple enough, but one must understand the child—its imperfectly developed tissues, its growth, and its peculiarities—to diagnose and treat its ills successfully.

Symptoms in infancy and early childhood are objective; consequently a keen, thorough, and minute observation is most essential to the physician. Frequently an intelligent mother or nurse notices many of the important symptoms and can recount the onset and course of the illness with a detail which is exceedingly valuable. Her narration should precede a systematically taken and complete case history. Under family history should be recorded the health of the parents, any acute or chronic disease they have suffered recently, and any disposition toward certain diseases or disorders. If dead, one should know the date and cause of death. Any family peculiarity or disease should be followed into the grandparents, and parents' brothers and sisters. Miscarriages and still births, if any, should be recorded, with dates and "possible causes." The presence of tuberculosis or its symptoms in the family or the patient's associates should be learned. Venereal disease, epilepsy, insanity, hemophilia, and malignant growths, if present in the family, have very important bearing. The number of living brothers and sisters of the patient should be recorded, with age, the present condition of health, an account of any recent or early illnesses. If any have died the date and cause of death should be ascertained.

The past medical history should begin with the parents. It is especially important to inquire into the condition of health of the mother at the time of conception, during the pregnancy, and at the time of birth. Any illness at this time, such as an eclamptic state, might have definite bearing on the child's condition. It should be learned whether or not the child was born at full term, and if premature, how much. The length and kind of labor are important, also whether or not it was necessary to use instruments. Should there have been any injury to the child, learn what it was, how extensive, and its effects. Ascertain whether or not the child was in apparent good health; whether or not the skin was clear and free from rash and desquamation; also whether or not the child cried at once, or required artificial means to establish respiration or overcome cyanosis.

It is then well to follow the growth and development of the patient, ascertaining its weight at birth, at six months, at one year, at eighteen months, and so on. If the mother has kept a record of weights its curve should be carefully studied and where any irregularities appeared an explanation should be sought. Measurements, if they have been made and recorded, are likewise valuable. The age at which the first tooth appeared, the regularity of eruption, and the completion of dentition, are quite helpful in determining the early state of health and nutrition.

Muscular development is learned from the age at which the infant first sat up, crept, stood, and walked. Mental development is shown by the age at which objects were followed with the eyes, objects and persons recognized, first words spoken, and first sentences formed.

It is at this time well to obtain a record of each illness or ailment the child has suffered, ascertaining in each case the date of onset, the possible cause, the duration, severity, recovery, and any complication or sequelæ. It is a good plan to begin at birth and follow up systematically to the present. Even then it is often necessary to ask if the child has had this or that contagious disease, has suffered from "colds," or if the baby seems to contract "colds" or bowel disturbances easily.

A most essential and valuable part of the child's history is the feeding. It is always most worthy of extreme care and detail, and should begin with birth, following each change up to the present diet. If the child was breast-fed, how long was breast feeding continued? Did it nurse from one or both breasts? How frequently was it nursed? Was it satisfied after nursing? Was it weighed before and after nursing to determine the amount of milk obtained? Did it regurgitate or vomit after feedings? What was the condition of the bowels—how many stools a day, and what was their color, consistency, and odor? Were there curds or "white lumps" in the stools, and if so, were they hard or soft? Did the stools seem slimy or contain mucus? Any blood, and if so was it light or dark, separate or mixed with the feces? Did the child gain regularly in weight, and how much?

The state of health of the nursing mother should be inquired into. Does she eat with a desire for food, or is the sight of it repulsive to her? Is she "stuffing" or does she eat regularly three meals a day? What is her diet? Does she drink plenty of water? Does she suffer from indigestion? Is she constipated? Is she worried, nervous, and worn out? Has she other children? Did she nurse them and how long? If discontinued, why? The condition of the mother has a direct effect upon the quantity and quality of her milk, and upon her nursling. A constipated mother has a constipated baby and very often to correct the mother's constipation corrects the condition in the baby.

If the breast feeding has been stopped, ascertain when and why. What was the baby given? What was used in the formula, and how was it made? What quantity was given at a feeding and at what intervals? Did the baby take the new food well? Did it regurgitate or vomit? Did it belch quantities of gas, or was there much flatus? What was the character of the stools and how frequent were they? Did the baby gain on this food? In this way obtain data on all foods given up to the present. Such a feeding history frequently points out the cause of a nutritional disturbance, and sometimes of other diseases as well. It will disclose the errors in feeding and be of vast aid in prescribing a diet.

Often the feeding history will include the present illness, especially in infants. It is essential, however, to get all details of the present complaint, beginning with the onset, following the course step by step or day by day with each development to the status praesens. How did the trouble begin? Was the patient in good health the day before the first "sign" of illness was noticed? Was there fever at the start? What was the temperature and what has it been since? Has the patient been drowsy and sleepy, or restless and wakeful? Has there been any cough, and if so, what type? Has respiration been rapid and labored, or has there been any nasal discharge? Has the appetite been poor? Has there been any vomiting or abdominal discomfort? What was the frequency and character of the bowel movements? Has there been plenty of urine, and what is its color? Does the child urinate frequently, or is micturition painful? Has the child assumed any unnatural positions? Has he put his hands to his ears or any other part of his body?

Such questions as these, taking up one system after another, make the possibility of overlooking valuable data less likely, and may stimulate the mother's memory and bring out forgotten symptoms.

A complete history makes way for a definite, clear-cut diagnosis, and especially is this true in pediatrics.

To obtain satisfactory physical examination of children is a matter of approaching and understanding them, that is, the ability to gain their confidence and determine quickly their mental attitudes. It requires considerable tact and frequently an enormous amount of patience, especially when the child is one who has been over-indulged by the parents. In such cases the examination may best be made in the parent's absence. Often the examination may be accomplished as an amusement to the child, but in the unruly and protesting patient a firm but gentle proceeding with the examination in spite of opposition may overcome the child's attitude and gain its interest and quiet. However, the experienced pediatrician may make a satisfactory examination although the child be protesting. The child wearies of long-drawn-out deliberation and handling, consequently a quick (not rushed), systematic and thorough procedure is essential with keen observation and skillful manipulation.

During the time taken in obtaining a history the physician has been observing the patient. He has noticed the expression of face and eyes, the posture, the state of nutrition, and possibly the gait. All the while he has "sized up" his patient and determined or entered upon his method of approach. Possibly he has observed the child without the child's knowledge—that is, he has apparently not taken any notice of his presence; or possibly he has gained the child's confidence by offering his stethoscope or some other article for the child to play with.

Should it happen that the patient be asleep, the child's position, the color of the skin, and the pulse and respiration rate and type should be noted before waking. Often abdominal palpation, cardiac and pulmonary auscultation, and general inspection, can be made without waking. Such an advantage should always be seized upon, and lastly percussion attempted—it, too, can frequently be accomplished while the patient is asleep.

For an examination the child should be stripped of all clothing and put in a warm, soft blanket. Most babies enjoy being relieved of their clothing and are in a happy state of mind. The proceeding should be systematic but it is most important to the success of the examination that its objectionable or disturbing parts be put off until all else is completed. That part of the examination which cannot be satisfactorily made during fretting or crying should be made early. Thus an abdominal palpation may be done before a cry makes the walls too tense—and cardiac auscultation should be finished while quiet prevails and before acceleration from excitement is brought on.

It is best to begin the examination with an inspection of the entire body. The facial expression is observed, the expression or movements of the eyes, the respiratory movement of the chest and abdomen, pulsations, the position the child assumes (its head, its body, its extremities), the general nutrition and development, the condition of the skin, the mucous membranes, the hair, the nails; in fact, everything to be seen on the surface. The position of the body may be only a habit, or it may be assumed to prevent or relieve discomfort. In pleural exudate the child lies on the side affected. In peritonitis the position of choice is on the back with the legs slightly flexed. In disease of the spine the child often lies on its face, while in meningitis the posture is on the side with the legs flexed and the head retracted.

Restlessness, the child tossing itself from place to place, is seen in the onset of infections, severe pains, (headache or earache) and in obstruction of the air passages. In rickets and spasmophilia it is a prominent symptom.

The eyes staring, eyelids wide apart and immobile, are characteristic of meningitis. Nystagmus or lateral movements of the eyes occur frequently in infancy. In the first one or two months it only indicates the undeveloped vision or muscle aberration, while later it is suggestive of partly obstructed vision, as corneal cloudiness or cataract, refraction error, or some retinal disturbance. It is usually seen in Spasmus Nutans and in amauroses or various cerebral disorders. If associated with general tremors and failure to hold the head upright, it suggests a multiple sclerosis.

The skin is one of the most sensitive and important organs in children. It is frequently an index of some defective function elsewhere, as well as of constitutional peculiarities. Therefore a most careful inspection of the skin is essential. Miliaria, sudamina, and intertrigo are often seen on the tender skin of infants. So too, both irritative and dietary eczemas. Blotchy or macular eruptions are seen in excessive sugar and acid dietary, excoriated buttocks in diarrheal conditions, and vesicles, pustules, and bleeding ulcerations from ammoniacal urine. Furuncular conditions are seen frequently in early life among the debilitated and neglected. Urticarial and diffuse or blotchy rashes arise in intestinal intoxications or protein sensitizations. Scabies and impetigo are common infections in pre-school and school children. The rashes of the exanthemata are, of course, best studied in childhood.

In tuberculous children the skin is often quite clear. It may be later dirty or weathered or it may be hairy. At times the characteristic tuberculid and syphilid is observed. In the cretin there is the harsh, dry, boggy skin, thick lips, short stubby hair, and impaired mental and physical condition. In rickets there is the pale, thin, waxy skin without edema, while in nephritis the skin is pale, waxy, and puffy.

In the new-born a slight yellow discoloration of the skin is normal, but persistence or deepening of the jaundice indicates some abnormal disorder of the blood, liver, or bile-ducts. Hemorrhagic conditions of the skin are seen in purpuric disease, in scurvy, and in injuries.

Old healed fissures in the muco-cutaneous areas about the mouth and anus are suggestive of syphilis.

Prominent venules are quite often seen in children and signify some obstruction to the returning cutaneous blood. In circulatory disturbances pulsations are noticed in the neck, in the precordium, chest, or epigastrium. They vary from a fluttering to a heaving type. One must also watch for retractions from respiratory disorders.

The subcutaneous tissue is well developed in infants and young children. It gives the body a full rounded contour and serves as a mechanical protector to the skeleton and as a physiologic one to the organs. In severe acute disease such as vomiting and diarrhea there is rapid wasting of this tissue. In chronic disease, such as tuberculosis, syphilis, and marasmus, the subcutaneous tissue has long since been used up, and the skin lies loosely and in folds over the skeleton. Excessive subcutaneous tissue (obesity) may be equally abnormal.

The lymph nodes, larger in the child than in the adult, are just palpable normally, but when they are more easily discernible they are pathologic. Chronic general enlargements are found in tuberculosis, syphilis, leukemia, and atrophy. Chronic localized enlargements may be tuberculous or sarcomatous. Acute hypertrophy from some local infection is frequently seen in children, and suppuration is common.

Muscular development is determined by the firmness to the touch and by the child's activity. Bony developments by the regular, well-formed skeleton. By inspection one discovers only the gross development of the child, but by measuring and weighing the more definite details are obtained. This part of the examination is best left until later, since it can be done with accuracy should the child be crying. Palpation, percussion, and auscultation are much more satisfactory when the child is quiet.

Respiration rate and character may be determined by inspecting the chest or by laying the hand on the epigastrium. During the first two years the respiratory movements are abdominal in type, gradually becoming costal by the fourth year. The rhythm and depth normally varies much, in infancy suggesting or simulating a Cheyen-Stokes type, which is of pathologic importance only after the second year. The respiratory rate is increased in the new-born, being about 45 a minute, decreasing gradually as the child grows, reaching 20 to 28 by the sixth year. It is also to be noted that respirations are markedly increased in all febrile attacks, so that we may often count 50 to 70 per minute at a temperature of 105 degrees. Such acceleration does not indicate pulmonary disease unless other symptoms such as dyspnea, wide movement of the alæ nasi, and expiratory grunt accompany it. However, should the normal ratio of the pulse to the respiration (1 to 4) be increased to 1 to 2, the cause of the rapid respiration is an affection of the lungs. Respiration with retraction of the intercostal spaces and epigastrium is seen in cases of obstruction such as laryngeal diphtheria.

Prominent costal breathing with groaning inspiration indicates impending cardiac collapse.

The thin chest wall of the infant quite often permits one to locate the apex beat of the heart by sight. In cardiac conditions a heaving impulse of the heart may be seen. Where the diseased heart has been hyperactive for some time the chest wall often shows a bulging cardiac area.

Inspection of the abdomen reveals contours and muscular action. Distended and prominent veins are seen over the right upper abdomen in enlargement of the liver. Bulging along the linea alba with respiration is seen in diastasis of the recti muscles.

To finish the general inspection it is necessary to observe the position in which the extremities are held, especially of the legs. In abdominal conditions the legs are flexed on the abdomen. This is also observed in meningeal disturbances. Tuberculosis of the hip or the lower spine has its characteristic position of the lower extremities. In scurvy the extremities are held in one position because of pain upon motion. Deformities should be looked for and the extremities compared for inequalities in bony or muscular development. These are noted in the various paralyses, either birth or acquired by disease.

The spine should be examined for curvature, either kyphotic, as in tuberculosis and rickets, or the scoliosis of rickets, or the compensating curvatures found in other conditions.

Up to this point it has scarcely been necessary to have laid hands on the patient. He has been undisturbed and possibly has remained quiet. Crying or fear has not been provoked and palpation of the abdomen can advantageously be done. The hand is laid flat on the abdomen and the resiliency of the walls is first determined. By gentle pressure rigidity of the muscles may be noticed and localized, if present. The lower edge of the liver can be palpated and outlined, and in children its lower border lies below the costal margin. The spleen normally can not be palpated. Other masses are abnormal. In tabes mesenterica, when the abdomen is flaccid, masses may be felt on deep pressure. In constipation fecal masses often are palpated. The abdominal rings and the umbilicus are examined for hernia, and the linea alba for diastasis of the recti muscle.

Abdominal palpation, if done skillfully, has not disturbed the child and crying has not been provoked. It is therefore advisable to listen to the heart sounds at this time. Auscultation is done with the stethoscope and with the same proceedure as in adults. Clearness, rate, regularity, and tone of the heart action are determined.

Irregularities are quite common in infants and children and do not have the pathologic significance which they have in adults. They are the result of the instability of the nervous control and to the irregularity of the respirations. It may be the definite juvenile type of irregularity. Other irregularities often occur after febrile attacks or in debilitating conditions. They are not of serious import.

Murmurs are more frequent in early life. Functional murmurs may occur, although the majority of cardiac murmurs occurring under two years are organic. Functional murmurs are always systolic in time and are heard best at the base. They are never very harsh nor loud, but are soft and blowing in type.

Organic murmurs before the fourth year are always suggestive of a congenital heart lesion. After the fourth year the usual mitral regurgitation, stenosis, and other lesions are as in later life. Congenital heart murmurs are various and the determination of the nature of the cardiac defect is extremely difficult, and at times impossible. It must be remembered that the aortic and pulmonary sounds in infancy are accentuated—much more so than in later life.

One may proceed now with the physical examination, following, of course, some definite routine. In this way the possibility of missing certain things in the examination is lessened. An obvious sequence would be the examination of the head, neck, chest, abdomen, genitals, and extremities.

The shape of the head must always be considered. At birth it may assume various shapes, caused by the pressure of its passage through the birth canal. These deformities disappear by the end of the first month. Most frequently the vertex is prominent and the head elongated or oval in shape. The anterior fontanelle at birth is open, and whether it gradually decreases in size from birth to its closure, or increases in size for a few months and then gradually decreases until entirely closed at the fourteenth to the twentieth month is still in dispute. Retarded closure of the fontanelle is most commonly indicative of a rachitic condition. It is marked in hydrocephalus. An early closure, that is, by the sixth month, is serious, suggesting an arrested cerebral development.

In a few days after birth the pressure deformities correct themselves and the head begins to assume a normal appearance. The misshapen head at birth may oftentimes be moulded by manipulation. The sutures are not as yet firm and the bones are loosely held together. This condition disappears rapidly after delivery. The opening in the outer table of the posterior fontanelle is obliterated by the sixth week.

At birth, the assymetrical head is due to pressure. If it occurs later it is due to rickets and to failure to change the position of the child as it lies in bed. The head should be examined as to the relative size of the cranium to the face, considering height as well as breadth. The frontal eminence should not be too square or prominent. The size of the anterior fontanelle should be noted and bulging or depression looked for. Bulging indicates an increased intracranial pressure, and depression a depletion as found in some chronic wasting diseases. Pressure over the cranial bones should be made to determine the firmness and absence of "denting," to exclude the papier-maché-like quality of cranium found in craniotabes. The size of the head should always be taken into consideration and definite measurements made. These are valuable only when compared to measurements of other parts of the body. This part of the examination, however, should be left until later, because it is not interfered with by the child's crying.

The absence of hair on the occiput, together with sweating of the head, is frequently seen in craniotabes. The skin of the head is a favorable location for numerous eruptions. Seborrhoeic eczema with crusts is very frequently seen in infancy, while in childhood irritations, crusts from eczema and parasites are met with. Furuncles and abscesses, with cellulitis of the scalp are frequent in infancy, especially in the malnutritic.

Tumors of the head occur frequently in the new-born. They are the doughy, edematous caput succedameum, the sharply defined, fluctuating blood tumor or cephalohematoma, and the cerebral hernia occurring at the location of sutures, pedunculated in form. These last two may contain only fluid or they may contain brain substance. The former fluctuates and is transparent, while the latter does not fluctuate, but may pulsate. Crying increases the size and tenseness of this tumor mass.

Examination of the eyes should include the position in the orbit, coordinate movements, and pupilary reaction to light and accommodation. In severe acute diseases with depletion the eye is frequently sunken in the orbit. This is noticeable to a greater degree in chronic wasting diseases such as malnutrition and tuberculosis. In cerebral disease, where there is increased pressure, the eyes are often bulging from the sockets. Eyes set widely apart, with the palpebral fissure slanting down and inward, are significant of Mongolian Idiocy. Incoordination of movements of the eye is normal in early infancy. So, too, is lateral nystagmus. Later they are indicative of some pathologic condition of the retina, cornea, or lens. They may also be of central origin. Ophthalmoscopic examination of the interior of the eye should always be made to determine the picture of the retina and choroid. Very frequently, in early tuberculosis, a tubercle of the choroid may be discovered and the diagnosis established.

Examination of the ear is made by inspecting the external ear for abnormalities, and of the drum by otoscope to determine the condition and position of the membrane.

The nares are examined either by reflected light or by direct illumination. As in adults, the position of the septum and turbinates, and the condition of the mucous membranes, should be noted. A membranous or profuse bloody nasal discharge is most likely diphtheritic. Persistent rhinitis and snuffles in infancy is very suggestive of syphilis.

Examination of the mouth should always be made last. It will probably provoke opposition and crying. The condition of the mucous membranes of the cheeks, the gums, the palate, and the throat should be noted. Disease of these mucous membranes is common in children. They are a favorite place for the manifestation of syphilis in the form of mucous patches. Simple stomatitis is quite common. Thrush, with its peculiar membranous covering, is often seen in neglected cases. Ulcerative stomatitis and noma are the most severe types of disease of this location. In school or institutional children, Vincent's infection of the gums is seen now and then. Thickening of the lips, with "drooling" persisting into childhood, is suggestive of idiocy. In the cretin the thickened lips and protruding tongue are observed.

The presence of a membranous exudate on the faucial tonsil and throat should always suggest the diagnosis of diphtheria. Smears and cultures should be made. The so-called Koplik's Spots are found on the mucous membrane of the inside of the cheek. Early in chicken pox a vesiculo-macular eruption is frequently seen on the mucous membrane of the soft palate. In scarlet fever is observed the so-called strawberry or raspberry tongue—bright red in color with the papillæ standing out prominently. Furring and coating of the tongue is seen in catarrhal conditions of the nose and throat, in digestive disturbances, and in febrile attacks.

The gums are examined for the alveolar ridge before dentition, and for the healthy mucous membranes. In scurvy one encounters the swollen red or purple hemorrhagic gums. They are spongy and bleed easily, especially after dentition has begun. A most minute examination should be made of the teeth. The first tooth erupts about the sixth month, and by the end of the thirtieth month all the first or milk teeth should be present. They should be regular in position and spacing. Discoloration and decay should be looked for and proper attention insisted upon. The permanent teeth begin eruption about the sixth year. Special attention should then be paid to the regular setting and the proper approximation of the jaws. At this time the severe deformities of the teeth and jaw seen later can be prevented or overcome with orthodontia. Very strict and regular attention to the teeth should be insisted upon.

Peg-like teeth, set far apart, with narrow notched edges, are very likely of syphilitic origin. Peg-like teeth with widely notched edges are probably of rachitic origin.

The faucial tonsil and its pillars should be carefully examined, the size of the tonsil and the condition of its follicles noted, and the presence of crypts or exudate looked for. Hypertrophied tonsils are frequently seen in childhood, but unless infected or of some mechanical obstruction, they are not operative. The small unsuspected tonsil is frequently the infected one, and an innocent looking tonsil may be imbedded and quite extensive in size and infection.

The condition of the pharynx should be carefully observed—where there is adenoid tissue mucus may be seen dropping down the posterior wall. In an examination one should always observe whether the child breathes through the nostrils or through the mouth. In early life the air passages are small as compared to those in later life. The anterior and posterior nares both are smaller, consequently there is more inflammation and infection of these passages. It may also be remarked here that the Eustachian tube in infancy is larger in caliber and shorter in length than in adults. On account of this condition, and the smaller air passages, otitis-media is so much more common.

One should always observe the frenum of the tongue to exclude the possible presence of tongue-tie. Other defects of the oral cavity, such as hare-lip and cleft palate, should always be looked for. A high narrow arch of the palate is found frequently in mental defectives.

The neck should be examined for adenitis, pulsating vessels, and muscle contractures. Also for rigidity of the muscles, which is indicative of meningeal irritation or disease.

The lymph nodes of the neck are common sites of acute hypertrophic and suppurative processes. These are usually secondary to disease of the mouth and throat. Tuberculous enlargement and breaking down of these lymph nodes is frequently seen in children.

In mumps the parotid glands are involved—possibly the submaxillaries may be sites of involvement.

Pulsating vessels are observed in severe heart or anemic conditions. Wry neck or torticollis may be due to a birth injury or it may be due to irritation from the underlying lymphatics. In the new-born there is usually a small tumor over the sterno-cleido-mastoid muscle, and the head is drawn toward the side affected. Special attention should be paid to the cervical vertebræ, as to position and mobility without pain. Tuberculosis of these vertebræ is not uncommon, though not so frequent as involvement of those lower down.

By the third month the infant should be able to hold his head upright. Inability to do this is suggestive of some form of infantile paralysis or idiocy.

Examination of the chest in children is performed by the same methods as in adults—that is, by inspection, palpation, percussion, auscultation, and measuring. As has already been noted, inspection is to determine the shape, development, and chest movements. The chest in infancy is round rather than the ovoid shape of adolescence.

Palpation is done to determine tactile fremitus, and the presence of enlargements or depressions not visible to the eye,—such as beading of the ribs in rickets. Enlargement of the mammæ with a slight milky-appearing secretion is commonly found in both male and female babies. It soon disappears but may show "caking" and at times suppuration.

Percussion is performed as in adults, but there are several things to be carefully followed—otherwise false notes will be obtained. It is necessary that the child be in a position in which the spine is straight and the shoulders level. If not, the note on the concave side of the vertebræ will be impaired and a false impression of the condition will be obtained. It is necessary that the muscles be relaxed—otherwise a false note will be obtained on the side of those contracted. It is also essential that percussion be done with a very light stroke, because of the thinness of the chest wall, the larger bronchioles and lesser alveolar air spaces, which cause harder strokes to produce false notes. Also vibrations, on account of the elasticity of the chest, are easily transferred to remote portions of the lung; or the impaired resonance of a circumscribed area may be entirely suppressed by the resonance of the healthy surrounding tissue. The resistance of the chest as felt to the applied finger is as important as the note obtained upon percussion.

During crying the percussion note is considerably dulled, unless the percussion be done at the time the child is taking a deep inspiration. Therefore only the notes obtained at the time when the child is inspiring are of value. The percussion note of one side of the chest should be compared to the note at the corresponding location on the other side. The borders of the lungs should be definitely determined and the extent of diaphragmatic movement obtained by percussing when the child takes a deep inspiration. This deep inspiration may be obtained in the older child by having it blow. In infants it is obtained during crying.

The heart area should be outlined on the chest wall. In infancy and up to the fourth year the left border of the heart will be located external to the nipple line; from the fourth to the seventh year most often on the nipple line; and from the seventh to the twelfth year within the nipple line. In infancy it will be found that this border is at least 1 cm. to the left, normally. If further to the left it is pathologic. The right border of the heart lies normally at the right edge of the sternum or a little to the left. The upper border approaches the second rib. Thus it is found that the apex beat in children is further to the left than in adults.

Auscultation of the lung is accomplished as in adults. In infancy it is often difficult by the continuous movement and crying of the patient. Very frequently it is impossible to use the stethoscope because of frightening the child. It is then sometimes advisable to auscult by the direct method, the child being held against the ear.

The most valuable information in auscultation of the lung is always obtained over the back. There is an area between the spine and the right scapula over which breath sounds are normally bronchial, and in examination this should always be kept in mind. It may also be noted that here the percussion note is impaired.

The breath sounds in early life are always more exaggerated than in adults. They are louder to the ausculting ear and expiration is much more clearly heard. This is so marked it is often spoken of as broncho-vesicular breathing, or puerile respiration. If on one side of the chest one finds an auscultory change it should not be presumed to be a pathologic condition until it is proved that it is not obtained at the same location on the opposite side of the chest. It is therefore most important that the sounds on one side be compared to those at corresponding location of the opposite side. The use of the stethoscope is often made less difficult if the child has been allowed to play with the instrument while the early part of the examination was being made. The application of the chest-piece, even though previously warmed, is made less startling to the child if the chest is touched first with the fingers.

Examination of the heart has already been completed, and it should be borne in mind that the systolic sound is stronger over the cardiac region than the diastolic one and that it is louder at the apex. In small children the second sound is louder in the pulmonary area and weaker in the aortic. The heart sounds in the infant are heard throughout the whole chest, but confusion in localization which might arise from this can be overcome by careful auscultation.

Congenital lesions of the heart must always be borne in mind and it must also be remembered that they are referred to the right side of the heart. These patients are prone to cyanosis and are characterized by muscular weakness and general malnutrition. Cardiac murmurs, if found in a patient who has any congenital malformation or defects, are very likely of congenital origin. To determine cardiac lesions the history is of great value—the infant with a congenital heart probably showed marked cyanosis in the early months of life. Increased heart activity or perhaps a murmur was heard in the first month, or probably the child was born in a state of asphyxiation. Such accompanying data is of much help in diagnosis.

Acquired heart lesions occur very rarely under the third or fourth year.

During the first one-half year of life the pulse rate is normally 120 to 140; during the second year 90 to 120; from the third to the seventh year 70 to 100. Acceleration of the pulse is very marked in crying, excitement, or fever. In order to obtain an accurate count it is frequently necessary to use the stethoscope, especially in infants and young children.

Examination of the abdomen should be made as to size and shape. Normally the anterior and lateral walls, when the child is in a recumbent position, are level with the lower margin of the chest. The sunken abdomen occurs in cases of malnutrition and depletion. The distended abdomen occurs in rickets, flatulence, and peritonitis. In Hirschspring's Disease the abdomen is very much enlarged, especially in the flanks and across the epigastrium. The other abdominal examination has been made previously.

In females the external genitals should be examined carefully for irritation or discharge. An adherent clitoris is frequently the cause of irritation. Gonorrheal vulvo-vaginitis is much more frequent than supposed. At times external examination may be negative, while examination of the cervix uteri, either by smear or inspection with illumination, discloses inflammation and pus. The hymen should be examined in each case to see that there is the usual small opening. If this is not present it will probably have to be made later at the onset of menstruation.

The male genitals should always be examined to see that the foreskin is loose and can readily be retracted. It should be observed that the meatus is open and at the proper location in the glans. Both testicles should be located in the scrotum. Often in infancy the testicles may lie above the scrotum, but their position is movable and they can easily be lowered. If the testicles are immobile in the canal, operative interference is necessary. One or both may be intra-abdominal. In such case the probability is that they will cause no trouble.

Examination of the anus should always be made. At this mucocutaneous border fissures or healed cracks—the rhagades of syphilis—manifest themselves. In the very young child or the child suffering from constipation it is always well that a rectal examination be made. A tight sphincter, as well as a fissure, is a frequent cause.

The extremities should be examined as to their musculature and bony development, and examination should be made over the femoral region for the presence of hernia or patent orifices. All reflexes should be tested for and compared one side to the other.

The spine should receive careful attention, process by process, to determine if all are present and that no vertebral herniæ exist. Tenderness or limited motion indicates some meningeal or vertebral disease. It must be remembered that in early life the spine does not have the normal anterolateral curvatures of the adult spine.

The infant or child should be weighed and measured to determine the physical growth and development. This should be done regularly and recorded so that comparisons may be made—those at one age with those of another. By such a record many disturbances can be detected.

Measurements of one part of the body are of value compared to those of another. Thus at birth one should find the whole body length four times the breadth of the shoulders—and twice the distance from the umbilicus to the top of the head or to the bottom of the feet. The head is one fourth the whole body length. The trunk is longer than the arm and the arm longer than the leg.

In the adult the shoulder breadth is less than one fourth of the body height and the mid point of the body height is at the symphysis pubes. The head is one eighth of the whole body height. The trunk is shorter than the arm and the arm is shorter than the leg.

The greatest circumference of the head in the new-born normally exceeds the circumference of the chest at the nipples by 1 to 2 cm.; and this equals or exceeds by 1 cm. the circumference of the abdomen. The abdominal measurement, however, is very unreliable on account of gaseous distention, and muscular contractions of the abdominal walls. The chest circumference should exceed one half the body length by 7 to 10 cm.

In hydrocephalus, idiocy, and rickets one finds either or both the head circumference and the cranial vault height increased. In microcephalic idiocy one finds a decrease.

Toward the end of the first year the circumference of the chest should equal the circumference of the head, and in the majority of healthy children the circumference of the chest exceeds the circumference of the head by this time or during the second year. It is to be noted that the earlier the chest measurement exceeds that of the head the more robust is the child. In early infancy the chest is round but by six months the transverse diameter has become the greater.

Weight is the best index to the infant's or child's nutrition. It should be recorded weekly for the first twelve months, monthly for the second and third years, and at least quarterly during the fourth, fifth, and sixth years—then twice a year. At birth the weight is about 7 pounds. Roughly, by the sixth month this is doubled, at one year trebled, at 2 years quadrupled, and by the seventh year 7 times the birth weight. For more definite details one should consult tables of weights and measurements.

The only reliable temperature of the infant and child is that taken by rectum. It can be done without disturbing the child if a little patience is used and the child not held too firmly. In fact it might be added here that in any examination of an infant or child the restriction of motion by holding increases the difficulty of the examination. Crying and excitement may cause a rise of 1 to 2 degrees F.

In examination of the nervous system one has to take into consideration in older children the subjective symptoms of dizziness, headache, variable sensations of pain, anesthesia, etc. Objective symptoms are the expression of the face, condition of the mental abilities, and the unconscious gestures. The gait and posture of the body should always be noted, muscular tone elicited, and degree of motion and strength observed. Tremors or convulsive movements should be watched and classified. The special senses should be tested as in adults.

No examination is complete without a urine analysis. Normal urine in children is the same as in adults, except in the new-born, when it is cloudy from mixture of mucus, epithelium, and uric acid. At this time, too, albumin and casts are physiologic.

As important as the examination of the urine is the examination of the feces. There is no difference in this examination from that of the adult. The limited diet of the infant and the specified diet of the child make correction of faulty digestion much more easy, and examination of the stools frequently determines the cause of many ailments.

A complete blood examination is always desirable. It must be remembered, however, that the hemoglobin varies from 65 per cent. to 100 per cent. in early life; that the red cells are increased to about 7,000,000 at birth and the white cells at this time 16,000 to 20,000. In childhood the white cells average 8000 to 13,000, and the red cells 4,000,000 to 5,000,000. In infancy and childhood the differential count differs in that the lymphocytes are increased to almost 50 per cent. as compared to 24 per cent. to 38 per cent. of adolescence. The eosinophils are normally increased 1 per cent. or 2 per cent. In the very young a few enucleated red cells are normal.

Blood pressure is less valuable in children than in adults. The diastolic pressure is 60 to 90 mm. while systolic pressure is 80 to 100. There is a gradual increase from infancy to puberty.

Radiography is quite an adjunct to diagnosis in children, not only of the bony structure but also of the heart, lungs, lymphatics, kidneys, and intestinal tract. The slow or obstructed passage of a bismuth meal in pyloric stenosis is readily followed by means of the flouroscopic screen, and movements of the heart and lungs, diaphragm and intestines, can be clearly observed and abnormalities detected.

Although the diagnosis of disease in early life may be attended with some difficulties or confusion they are in great part overcome by a complete history and a thorough detailed examination. It is in pediatrics probably more than in any other branch of medicine, that keen observation and inspection, proper interpretation of symptoms, and quick, skillful, and thorough examinations are most essential.

The cry of the infant is typical in different conditions and an appreciation of the type is of diagnostic value. In colic there is a violent paroxysmal crying, frequently repeated with a renewed vigor. The infant strains and draws the thighs up on the body. The cry stops only temporarily upon taking food. It ends upon the expulsion of gas. In injury the cry is violent but soon stops upon diversion of the child. In hunger there is a fretful and persistent cry, appeased by food or drink. In pain such as earache, the cry is shrill and persistent with violent outcries, and not stopped by food. Skin irritation causes an obstinate cry. In pneumonia the cry is held back on account of pleurisy, and instead, a grunt, with often a sharp cry after coughing. In laryngitis the cry is hoarse, and inaudible in stenosis of the larynx. There is a sudden shrieking and moaning type of cry in meningitis. In diseases of the bones and in dentition there is the sudden outcry during sleep. In scurvy the child shrieks in pain on being handled. In anger the child cries violently, stiffens itself, and if old enough stamps its feet or throws itself on the floor or bed. An overindulged child cries in anger but stops immediately when attention is given it.

Cough in early life is very similar to that of adults. In pharyngitis there is the hacking cough from a tickling sensation, worse when lying down; in pneumonia the suppressed cough; in laryngitis and tracheitis, or where there is a pressure upon the bronchi, as in tracheo-bronchial lymphatic enlargement, there is the tight hoarse cough. The cough of bronchitis is dry at first, then loose and rattling when the secretion is increased. In asthma the cough is usually short with wheezing—dry at first, then loose. In whooping-cough there is the paroxysmal attack increasing in violence and ending with an inspiratory whoop.

Vomiting is a most frequent symptom in infants and children. It comes on at times with the slightest provocation, the nausea being of short duration and soon followed by a complete emptying of the stomach. The child is pale, exhausted, and in a cold perspiration, The onset of acute febrile disease is frequently first manifested by vomiting. The regurgitation so frequently seen in infancy is due to an unestablished action of the cardiac orifice of the stomach or to a food too high in fat or carbohydrate. Persistent projectile vomiting immediately after feedings in infants suggests pylorospasm or pyloric stenosis. In toxic conditions, as from food intoxication, the vomiting is severe, prolonged, and with much retching. Recurrent vomiting is most distressing and due to toxic or acid absorption. Vomiting repeated over several days suggests meningeal disorder, while vomiting accompanied with constipation or mucus stools, followed later by vomiting which is "fecal" in type, is diagnostic of intestinal obstruction. In peritonitis there is this same vomiting with constipation and no peristalsis. The vomiting of acute gastro-intestinal disturbance is of short duration and accompanied by diarrhea. Hypertrophied tonsils are also a frequent cause of vomiting.

Diarrhea, so frequently a symptom in early life, is usually brought on by irritation from improper food. It may be of toxic origin, or it may be an acute or chronic infection of the intestinal mucosa. In dentition, or in any condition in which the infant's unstable nervous system is temporarily deranged, one often finds diarrhea. The stools are loose or watery, frequent, the odor and color reaction varying according to the fat, carbohydrate, or protein indigestion. Bright blood in the stool is from a lesion low down in the bowel, such as fissure, polyp, or irritation. If bloody mucus it may be from intussusception, ileocolitis, or one of the hemorrhagic diseases. In the new-born it may be melena. White lumps or masses are usually fat or protein curds—microscopic examination will differentiate them. Foamy or frothy acid stools are from carbohydrate indigestion. Small brown and green fecal stools are seen in starvation. Certain drugs will cause discoloration of the stools.

Constipation is a common symptom at this period of life. When occurring in the breast-fed the mother is generally so afflicted and every effort should be made to correct this condition in her as it undoubtedly has some effect on the nursing infant. Other causes are too little fluid intake, too high a percentage of fat or protein, or too low a percentage of carbohydrates. It may be from an atonic condition of the bowel and a lowered general condition of health. Frequently constipation is the result of anal fissure or injury. Repeated enemata or suppositories will invariably cause constipation by overdistending the colon or by doing injury to the rectal or anal mucosa or muscle.

The child is very prone to disorders of the nervous system. Over excitement or exertion, or improper feeding, cause restless sleep or night terrors. General twitching or convulsions, or wild excitement and delirium, frequently usher in any acute febrile attack; they also frequently accompany gastro-intestinal disturbances, especially the intoxications. Any peripheral irritation may cause these attacks. A few babies suffer convulsions with the eruption of each tooth. These children, who upon the slightest disturbance develop convulsions, have undoubtedly inherited a neurotic, hysterical predisposition, which may very likely persist through later life in one or another form of neuropathy.

In tetany one sees attacks of muscle contraction, especially of the extremities and of the larynx. Convulsions and laryngospasm are common. The nervous system is extremely sensitive to both mechanical and electric stimulation. Frequently these are very slight or "latent" in manifestation. Rachitic babies are very subject to tetany and the same nervous manifestations. Convulsions and attacks of asphyxia are the most frequent symptoms of status lymphaticus. Similar attacks to these are seen in cases of protein anaphylaxis.

At the onset of an acute illness the child may be extremely restless, toss from side to side of the crib, or demand, in succession to be taken up, put into bed, carried or rocked. It is quickly dissatisfied with each position and so extremely excited that no posture gives it apparent ease or comfort. If toxic the child is quite still and lies in bed, drowsy or asleep, apathetic to all surroundings, and restless or fretful only when disturbed. This is frequently seen at the beginning of an infectious disease. After exhaustion or illness of long duration, the child may lie for hours in one position unobservant of all surroundings.

Restless sleep in early life is observed in rickets, indigestion, dentition, hunger, and the nervousness from over exertion or excitement. Frequently it comes from the child being too warm—or from "colds" obstructing respiration—or from hypertrophied tonsils and adenoids. It is present in the acute inflammations such as otitis, stomatitis, and tonsilitis.

Lying on the side with the head drawn back and body rigid or hyperextended and extremities flexed is indicative of meningeal irritation or inflammation. To some degree this position is seen in the marantic baby. Lying on one side may suggest a pleural involvement of that side, or it may be because of earache, to have the affected ear against the warm pillow. In colic the infant writhes in pain, paroxysmally flexing or extending the body, holding the abdominal muscles tense in straining, and the thighs flexed closely upon the abdomen. In scurvy the infant lies quietly, not attempting any motion because of the incurred pain. In tuberculosis of the bones sleep is interrupted by sudden outcries and the part is held fixed because of pain on motion. In fracture, dislocation, and osteitis there is no voluntary motion of the part involved.

Inability to hold the head erect and reluctance to move the extremities may be due to general weakness, to a central nervous disorder or infection, such as poliomyelitis, or to rickets or syphilis.

Extreme restlessness and incoordinate jerking movements are seen in chorea. To and fro motion of the body while in a sitting posture is suggestive of masturbation.

The child's expression in severe illness varies from one of extreme agitation, at times maniacal, to one of extreme apathy or the unconsciousness of a toxic state. In milder illnesses it varies from one of excitement to one of drowsy disconcern. The eyes may be constantly moving, staring, dull, or closed. In atrophic conditions or after long illness the eyes may be only partially closed in sleep. In some acute illnesses and toxic conditions the eyes are sunken and deep rings encircle them. This is also noticed after prolonged sickness. The eyes are rolled up or out in convulsions. One or both eyes may be crossed in intracranial disease, or from a congenital defect.

Nystagmus may be caused by a central lesion, as chronic hydrocephalus or meningitis, a lesion of the fundus oculi, or by a functional disturbance coupled with a nodding or rotary spasm of the head. It is present in Frederick's ataxia. In reflex irritations as in dentition one may find nystagmus; also in general weakened condition. In hydrocephalus the eyes are turned down and the sclera above the iris is visible.

Photophobia is noticed in disease of the eye itself, in other diseases in which the eye is secondarily involved, (as in measles), and in meningitis. The child, to keep out the light, will cover the eyes with the hands or bury its face in the pillow or bedclothing.

A child suffering from hypertrophied adenoids has a dull, stupid expression. The mouth is held open and mucus or pus is constantly blocking the anterior nares, ofttimes producing excoriation of the upper lip. This same condition, combined with enlarged lymph nodes of the body, especially those of the neck, and with rickets, is indicative of exudative diathesis. If the lymphatics are suppurating, the condition is the one formerly termed scrofula.

Bloody mucopus from the nose is probably diphtheritic. Persistent nasal discharge in the infant suggests syphilis.

Marked movements of the alæ of the nose with respiration are observed in pneumonia and in laryngeal obstruction. The facial expression is strained and anxious.

Facial grimaces during sleep suggest nervousness, gastro-intestinal disturbances, or some slight pain.

In the new-born baby the skin is red to a pink with a more or less yellow cast. This jaundice may be very extreme and of a serious prognosis. Maceration of the skin at birth, especially of palms of hands and soles of feet, is most suggestive of lues. Many irritations and inflammatory skin lesions may be present at this early age, when the skin is so soft and tender. They may be from the simple dermatitis, intertrigo, etc., to the extreme type of pemphigus and dermatitis exfoliative. In congenital heart disease there is very marked blueness of the face, fingers, and toes. Such a condition arising acutely might be seen in laryngeal obstruction or in pneumonia.

Infants are commonly afflicted by eczema caused by faulty feeding —either too high fat or sugar content. These infants are likely to be sensitive to food proteins.

Urticarial eruptions are very commonly seen in early life and differ because of frequent associated papules, vesicles, or pustules. These may be from sensitization to one or more vegetable, animal, or bacterial proteins, or due to certain fruit acids and their resulting fermentation. Any disorder in the digestive tract or any nervous derangement may cause urticaria. Parasitic diseases of the skin are very common and especially after the infant age. All lesions are exaggerated in children and pustulation is a quite common complication.

Exanthemata are most common in childhood, hence the different eruptions are best seen at this time. In acute intestinal intoxication there is often seen a diffuse punctate rash resembling the scarlet fever eruption or the maculo-papular rash resembling measles. There is no desquamation following it.

Visible dilated veins of the scalp are seen in rickets and more marked in hydrocephalus; of the chest in severe dyspnoeic conditions; and of the abdomen in peritoneal disease, as tabes mesenterica—tumor growth of the abdominal organs—and in peritonitis.

The skin is pale and clear in tuberculosis. In nephritis, rickets, heart disease, and depleting diseases there is marked pallor of the skin. In rickets profuse perspiration of the head is present. In syphilis the skin has a "weathered" discoloration and often in severe enteritis the same muddy brown color is seen. There are cracks or fissures of the skin or mucous membranes about the mouth and anus in syphilis. There may also be noticed a copper to brown colored maculo-pustular eruption known as syphiloderm over the lower part of the trunk and extremities.

Cold hands and feet are found in cases of poor circulation, and coldness of the part affected is seen in poliomyelitis.

The skin has a subnormal temperature and a bogginess in the cretin. Edema is present in cardiac and renal disease. It may often be seen in the marantic baby that is fed high percentages of protein.

Club fingers are seen in chronic lung disease and in congenital heart cases. Chronic swellings of the shafts of the phalanges are sometimes tuberculous or syphilitic. Swellings of the joints are seen in the various forms of arthritis. Nodular masses on the shafts of the long bones are seen in syphilis and scurvy. Antero posterior curvature of the spine is caused by rickets (a bowing), or by tuberculosis (a knuckling). Lateral curvatures are due to rickets, to faulty posture, to unequal extremities, or to long-standing pleuritis. Various spinal curvatures are present in paralyses affecting the trunk muscles or those from the trunk to the extremities.

The mucous membrane of the mouth and lips is a vivid red in the marantic infant. In congenital lues it is frequently involved as by fissures and mucous patches. Bluish red areas with white centers are the Koplik spots of measles, and are present on the oral mucous membranes preceding the rash. Vesicles of the mucous membrane of the soft palate are seen in early chicken pox. A red swollen Stenson's Duct orifice is seen early in mumps. In scarlet fever the fauces are intensely swollen and bright scarlet in color. Later the light colored prominent papillæ with bright red mucous membrane about them make the "strawberry tongue." In whooping cough the mucous membrane is a bluish red and sublingual ulcerations are frequently found.

A coated tongue with foul breath may be due to some digestive disorder, or to a catarrhal or diseased condition of the nose and throat. In acidosis there is a fruity odor to the breath, while in gastro-intestinal disturbances with vomiting there is a distinctly acid, sour odor.

The gums in scurvy are a purple, swollen, spongy mass. The frequent gum boil of childhood is usually an alveolar abscess arising from a neglected or diseased tooth. The permanent upper incisor teeth are often notched in congenital syphilis. A similar condition is sometimes seen in rickets but the notching is more rounded. Peg shaped and irregularly set permanent teeth are also indicative of congenital lues. Decayed teeth are frequently the foci of infection in arthritis, chorea, endocarditis, nephritis, and various other serious ailments. Therefore dental hygiene is most essential in early life.

The control of the thermic center is poorly developed and heat absorption and dissipation by the skin is greater in infancy. The latter is because of the greater skin surface per pound of body weight of the infant as compared to that of the adult. Consequently temperature variation is more marked in early life, and high elevations are more common even in the slightest of disturbances. Intestinal toxemia, infection, and heat stroke cause very high temperature. Slight elevation may be seen in children of run down condition and in those convalescing from an acute illness. External heat (as hot water bottle) frequently causes a fever temperature; cold will readily cause a subnormal one. This is especially true in thin, wasted babies. Subnormal temperatures are frequently seen in marasmus, shock, collapse, hemorrhage, or in the premature. A normal temperature by rectum may show a variation from 98 to 100 degrees F.

The pulse rate normally is higher in infancy than in childhood and adult life. The normal rate during the first six months of life is 120 to 140; during the second six months it is 105 to 120; from the second to the sixth year, 90 to 105; from the seventh to the tenth year, 80 to 90; and from the eleventh to the fourteenth year, 75 to 85. Exercise, excitement, and fever cause much more acceleration of the pulse in the young than in the adult, and unless the pulse rate is counted while the child is perfectly quiet or asleep it is of little value. Irregularities are present frequently and more especially in the younger infants. These irregularities are natural and are present even in sleep. They are, however, more accentuated and more marked after any illness. Comparatively the increase of pulse rate per degree of temperature is less in infancy than in the adult. Instead of ten beats for each degree of temperature increase it is about four.

Respiration varies greatly in infancy. The rate is increased by the slightest fretting, excitement, or fever. There is also more or less irregularity of rate and depth; at times even a Cheyne-Stokes type is seen during the sleep of normal infants. A regular rythm of respiration is not established until the third year. Dyspnea is frequently seen in upper respiratory tract infections or occlusion from any cause, and in pneumonia. In the latter there is also a grunt at the end of expiration. In acidosis, uremia, severe anemic conditions and heart diseases there is dyspnea, the intensity varying with the severity of the underlying condition. Respiration is diaphragmatic in infancy, and costodiaphragmatic in childhood. The average normal rate at birth is 35 per minute; at one year, 27 per minute; at two years, 25 per minute; at six years, 22 per minute; at twelve years, 20 per minute. Respiration is higher pitched and harsher in infancy, becoming less so as adult life is approached. The normal respiration is bronchovesicular in early life as compared to the vesicular of the adult. This is because of the thinness of the chest wall, more bronchial air space, less alveolar air space, and shallow respiration. Bronchial breathing is diagnosed with difficulty in infancy because of confusion with the puerile respiration. In infancy bronchial respiration is not dependent upon loudness, harshness, or pitch, but upon prolongation of expiration, if elicited, the "nearness" of the sound, and the nasal whir or blowing quality that it presents. The respiratory murmur is fainter over the involved lung.

Normally the ratio between respiration and pulse is 4 to 1, but in pneumonia the ratio is 2½ or 2 to 1.

The prognosis of disease in infancy and childhood depends upon the age of the patient—the younger the child the more dangerous the disease. Mortality is greatest the first day of the first week, the first week of the first month, the first month of the first year, and the first year of childhood. The death rate declines from birth to the fifteenth year of life.

The new-born child, with an unstabilized nervous sytem, is brought suddenly into a new environment to which it must gradually adapt itself. Its respiratory, circulatory, and digestive systems are for the first time brought into use, consequently disease or disorder finds easy entry should proper care, hygiene, and feeding be neglected. It may be said, however, that should the child be born full term with no defects of structure, no congenital disorders, and no birth injuries, its reaction to an acquired disease is probably as good as that of an older infant. In many conditions the infant's rapid metabolism offers most remarkable means of repair.

The high mortality of the new-born is caused from premature birth with congenital weakness; from injuries received in passage through the birth canal; from pneumonia, atelectas is, syphilis, defects in development, asphyxia, and accident.

The high mortality during the first year of life is due to acute gastroenteric disorders, which are largely preventable, but to which is attributable the largest percentage of deaths at this age. The deaths ascribed to convulsions and marasmus are to a great extent of gastro-intestinal origin, and should be classed as a sub-division of this cause. The other causes in the order of their importance are permaturity or congenital weakness, acute respiratory diseases, congenital malformations, acute infections, especially pertussis, tuberculosis, and syphilis. In the summer months deaths from gastro-intestinal diseases are highest, while in the winter the greater number are from respiratory disorders.

Sudden death so frequently seen in atrophic infants is probably due to cardiac collapse, lowered resistance, and nervous instability. Radical changes of weather are always most dangerous to them. It often happens that these malnutritic babies, showing no obvious change of condition, are found dead. The nurse may have completed feeding the infant, tucked it into its crib, gone to rinse the bottle and nipple, and return in a few minutes to find the baby dead. In older children of apparent good health, sudden death is probably due to status lymphaticus, a general lymphatic overgrowth, especially of the thymus.

During the second year, gastro-enteritis is again the most important factor of death, and the acute respiratory disorders come second. The contagions become more common, measles, diphtheria, pertussis, and scarlet fever, in the order named, are the outstanding causes of death. Tuberculosis and meningitis, too, appear more frequently.

From the age of two to five years deaths more commonly occur from the acute infections, acute respiratory diseases, tuberculosis, and meningitis. Diphtheria and scarlet fever are the most prominent of the infections, measles and pertussis showing less fatalities, but their complications and sequelæ make them still quite serious.

It must be remembered always that young children fail to localize pain, consequently, unless complete examinations are made, otitis, mastoiditis, appendicitis, or duodenal ulcer may be overlooked.

Doubtless many of these involvements are not recognized and deaths are attributed to other causes. Prompt discovery of such conditions would change the prognosis quite decidedly.

The smallest mortality exists from the fifth to the fifteenth year. Diphtheria, scarlet fever, and acute respiratory diseases become the leading causes of death, while diseases of the meninges, the bones, the heart, and the appendix are less prominent.

During the last few years the frightful loss of infant life has been markedly reduced by the splendid work of Baby Welfare Organizations. Their teaching of prenatal, infant, and child care has shown wonderful results, as well as the establishment of better milk and food supply, insuring proper feeding and hygiene.

The mortality of children has been greatly reduced by diphtheria antitoxin, typhoid vaccines, and anti-meningococcic serum. Toxin-antitoxin prophylaxis gives promise of reducing diphtheria fatalities.

In the treatment of diseases of children medication should be at a minimum and of the simplest form. Far too often is the ill child overly and unnecessarily drugged. Such treatment not only is repugnant to the child but excites its aversion to any medication. It arouses the child's suspicion of every spoon or glass of food or fluid offered, and results in a refusal to eat or drink. This does not mean medical nihilism. It does mean that the medication be only that specifically and definitely indicated, that it be of small dosage, and so far as possible, palatable. With such a minimum and agreeable medication coupled to intelligent nursing, indicated general and local treatment, proper diet and hygiene, all cases invariably do best.

The physician should write out in detail all instructions, designating the time and dose of medicines, the time and preparation of local or general physical measures such as counterirritants, cold or hot applications, sponges, or baths; the time, amount, and preparation of the articles of diet; the clothing of the patient, and the proper ventilation and temperature of the sick room.

Children bear certain medications especially well, while others are poorly borne. Of those which are well taken may be mentioned the alkalin salts, antipyrin, alcohol, belladonna, the bromids, chloral, the iodids, the mercurials, strychnin, and in small doses some of the opiates. Morphia, cocain and heroin are to be used cautiously in the treatment of children. The salicylates, the acids, and the astringent preparations of iron cause gastric irritation. The antipyretics are seldom used in early life, and only when the nervous symptoms from temperature cannot be controlled by cold applications, sponges, and other external means. Phenacetin and antipyrin are the most serviceable.

An enema or colon flushing with tepid saline or soda-bicarbonate solution will frequently lower a very high temperature and relieve nervous twitching.

Bowel astringents are used less and less, and more dependance is placed on proper diet. However, bismuth, either subnitrate or subcarbonate, and the mistura creta, are occasionally used to soothe. In certain cases of ileo-colitis accompanied by great tenesmus, laudanum in raw starch solution given by rectum often proves of good service.

Sedatives are quite well borne and are frequently used. The bromids especially are of value in restlessness, sleeplessness, colic of infancy, and cough. In convulsions larger doses are required and for quicker effect it is well combined with chloral. The latter drug is somewhat irritating to the stomach, consequently it is given per rectum in double dosage. Antipyrin is also much used, both in convulsions and in croupy laryngeal coughs to relieve the spasm.

Belladonna is well borne by most infants and children, and in comparatively larger doses than in adults. In combination with bromid and antipyrin the resultant action seems very desirable in irritative conditions of the respiratory, alimentary, and urinary tracts.

Opium is used in infancy and childhood comparatively infrequently and always with caution. However, in proper dosage it may be used with excellent results. It is valuable in treatment of harassing coughs and in pneumonia to combat fatigue. Paregoric, laudanum, Dover's powder, codeine, and morphia are those forms most used.

Stimulants are quite essential in the treatment of diseases of children, but they should be used only when indicated and not throughout an illness lest the effect be lost by the establishment of a tolerance. Those most used are adrenalin, aromatic spirits of ammonia, carbonate of ammonia, alcohol in form of whiskey, caffe in, camphor, digitalis, and strychnin. Adrenalin, caffein, and camphor are best given hypodermatically. Digitalis and strychnia may also be given this way when quick action is desired.

The tonics particularly adapted to children are nux vomica, iron, cod-liver oil, and malt. Nux vomica is most useful in convalescence and may be advantageously prescribed in sherry wine. Iron may be given in the form of bitter or sweet wine, the saccharated carbonate, or as in Blaud's pill in older children. Basham's mixture is of value, and arsenic in the form of Fowler's solution may serve a good purpose. Iron citrate hypodermatically is very efficient in severe anemia. Frequently iron medication is overdone, and prescribed when a simple alkalin would be quite or even more efficient. Hypodermic medication in early life is less used than in adults because of the pain and fright it occasions. However, in respiratory or cardiac depression it is invaluable.

Cod liver oil is most valuable in convalescence from diseases of the respiratory tract, in anemia, and especially in rickets. It is often taken very well as the plain oil, though it may be given as an emulsion. Cod liver oil is not well taken after digestive disorders and should be avoided in cases in which there is a fat disturbance. During the hot weather cod liver oil is not well borne.

Malt is taken advantageously as a reconstructive tonic and can be given when cod liver oil is contra-indicated. The malt and cod liver oil combined as an emulsion is well taken and efficient.

Beyond all doubt there is no part of therapeutics so greatly and persistently abused as are laxatives, cathartics, and purges. Laxatives are often given as a part of the daily routine and cathartics and purges with the regularity of the old time Saturday night bath. The child's digestive system has scarcely time to recover from the one dose until it is time to take another. If proper attention were paid to the selection of the diet, regular habit of evacuation and sufficient physical exercise, this pernicious drugging could be stopped. It might be added also that the pre-operative catharsis, purge, and enemata, are undoubtedly unnecessary and harmful. They annoy, weaken, and deplete the patient just at the time when all his strength and vitality is needed. A more judicious use would be better. The use of these drugs would then be limited to sickness in which elimination or thorough cleansing out of an irritant might be indicated. Milk of magnesia is taken well by children and acts well as a mild laxative. If greater effect is desired the dose repeated in one hour does well, or aromatic fluid extract of cascara might advantageously be added. Castor oil seems to act best when quick, complete cleansing of the bowel is desired. It may be given alone or combined with syrup of rhubarb. Occasionally a child cannot retain the oil. It is then better to resort to calomel in divided doses followed by milk of magnesia, or in older children the citrate of magnesia. The repeated doses of milk of magnesia do well too where oil is vomited. Calomel is well borne and complete in action, though slower. It may be followed advantageously by castor oil.

In babies depleted severely by diarrhea, or atrophic infants weakened greatly by acute disease, drastic purgation should not be attempted, in fear the little remaining vitality be wiped out. Milder measures will be efficient.

The daily or frequent use of enemata or suppositories should be discouraged because of the habit they produce, and because sooner or later they will do injury to the anal mucosa. It is quite often one sees cases of an atonic sigmoid which was produced through the daily ballooning up of that part of the bowel by large enemata; and it is just as frequently one finds constipation due to any injury of the anal mucosa or to an established habit of anal stimulation.

Medication by enemata or suppository is useful when the stomach is intolerant or when the drug used is irritating to the stomach. Chloral and bromids are thus frequently administered in watery or starch solution. Opium in the form of laudanum is also given by bowel in starch solution, both for its local and general effect.

Nutrient enemata are less used than formerly. The bowel too soon becomes irritated and the fluids are expelled. For this reason the drop method of enteroclysis is of less use in infancy. The tube, a foreign body, irritates, and expulsive peristalsis is soon set up. It is better to give the determined amount of fluid at one time, repeating at prolonged intervals. The buttocks must be elevated and pressed together for one-half hour following the administration of the fluid.

Colon irrigations are practiced much in children to cleanse the lower bowel of fecal masses or mucus. They are particularly beneficial also in reducing a high temperature. Frequently-repeated irrigations produce irritation.

Hypodermoclysis of normal saline solution is a most valuable means of supplying fluid to the depleted or toxic infant, as in cases of severe diarrhea and intoxication, or when fluid is not retained by the stomach or bowel. The injection may be made of certain amounts at intervals, or may be given more slowly and continuously. The preferable sites for subcutaneous injection are the back between the scapulæ, or the abdomen, because of their large amount of areolar tissue.

Larger amounts of fluid may be given quite safely into the peritoneal cavity. Absorption is more rapid and the painful areas at the sites of hypodermoclyses are avoided. With ordinary precautions danger of carrying in infection or of puncture injury to the bowel is very small. The fluids so added to the tissues act quickly as a circulatory stimulant, and a known amount is definitely given. It is especially useful in infancy because of many depleting conditions and the great number of children whose stomachs are easily irritated. The introduction of fluid by this means is frequently delayed too long.

Intravenous injections are used in babies with good results. Salines or medicated solutions are given into the longitudinal sinus or the external jugular vein in infancy, and into any of the superficial veins of the neck or arms of older children. Collapse is so treated with good results. In severe malnutrition or acidosis glucose solution is frequently given. The neutralized Salvarsan is given intravenously in the treatment of syphilis.

Blood transfusion is indicated in severe hemorrhage, anemia, or the hemorrhagic diseases of the new-born. In the persistent oozing hemorrhagic diseases the intramuscular injection of 20 c.c. to 30 c.c. of whole blood freshly drawn from one of the parents produces wonderful results. This procedure, being simple and easily done, is well to keep in mind.

Diphtheria antitoxin has done much to reduce the mortality rate in children. It should be given at once should diphtheria be suspected. Given intramuscularly it gives almost as quick results as the intravenous injection, and is accompanied with less shock. The Shick test and administration of toxin-antitoxin to immunize gives promise of far-reaching good. The reaction of all children should be determined and those immunized who require it.

In the very early stage of epidemic meningitis, intravenous or intramuscular injections of the antimeningococcic serum are indicated —preferably the intravenous. Later the injections are intraventricular or intraspinous.

Autogenous vaccines should be used whenever possible. By their judicious use much good can be accomplished. Staphylococcic vaccines are of value in the furunculosis so frequently seen in malnutritic babies. Streptococcic vaccines are doubtfully satisfactory. In pneumonia the pneumococcic vaccine is rather disappointing, although there seem to be some good results in Type I infections. In vulvovaginitis the gonococcic vaccine has not been of any marked satisfaction. The colon bacillus infection in pyelitis in certain instances may be controlled by vaccine therapy, but cured only when it is used in conjunction with other treatment. Typhoid vaccination as prophylaxis is as much indicated in children as in adults. Tetanus antitoxin has greatly reduced the terrible suffering and loss of life after injuries. Failure to use it in the first treatment of open wounds from injuries should be classified as criminal neglect.

Mustard is probably the best counterirritant for use with children, although camphorated oil is very much used but less reliable. In local conditions the mustard paste is best. It is made of one part mustard to four or six parts wheat flour, mixed with warm water and applied between layers of gauze or muslin only long enough to produce redness. The best results from mustard plaster in pneumonia are obtained in the very early stage—later it is of doubtful benefit. The plaster should never envelop the whole chest. By so doing the movements of the chest are limited and the sore lung has an additional hindrance. General counterirritating effect is best obtained by immersing the whole body in a bath of warm water containing mustard, one tablespoonful to the gallon. This is most useful in convulsions, cerebral congestion, or in collapse and prostration. The mustard pack is less effective but is not so disturbing to the child. A sheet is wrung out of warm mustard water, wrapped about the child who is then covered with a blanket. Fifteen or twenty minutes of this causes a good red reaction of the skin.

Dry cups are only occasionally used in children and wet cups never.

Heat is always more agreeable to the child than cold. When cold is indicated it should be applied gradually, especially in small atrophic or weakened infants. There should be some protection between an ice bag and the thinly covered brain of an infant.

In ophthalmia compresses should be kept cold by changing every few minutes.

Hot packs, hot baths, and vapor baths are of value with indications similar to those of adults.

In the many eczemas and irritations of the tender skin of the child the bran bath is of great value.

A quick cold sponge or spray is a wonderful tonic to all infants and children if they react. Even the atrophic infant's circulation is improved by them. It should be given after a warm bath and in a warm room. Should the child be blue and pinched afterward he does not react and the water should be warmer.

Nasal irrigation with a saline solution is sometimes necessary in infants and children but it should always be done with great care on account of forcing infection into the Eustachian tube and middle ear. In young children and infants nasal sprays are little used. Solutions of silver salts or oily medicated solutions are dropped into the nostril and allowed to run back by gravity. Sprays are often used for cleansing the mouth and throat, but for the treatment of infection the swab is preferable.

Hoarseness, croup, and cough in children are well treated by inhalations of medicated steam. A small room may be filled with steam and the child be kept in it, or a tent may be erected over the crib and steam run into it from a kettle.

Gastric lavage is frequently done in infants and young children suffering from acute or chronic gastric indigestion. In the treatment of dilated stomach, pyloric stenosis, and poisoning it is of great value. The tube is easily introduced in infants, but in older children, who are frightened and struggle so much, it is most difficult. Water or saline solution (100°F.) are most used. The results from washing out the stomach are most satisfactory.

Gavage or the introduction of food into the stomach through a tube is resorted to when an infant is unable to swallow sufficient for its nourishment, as in prematurity or extreme malnutrition; when food is refused; when there is persistent vomiting; and in delirium or coma. It is an easy procedure in infancy and causes very little disturbance. Nasal feeding is used in older children who will not permit gavage, in cases of intubation or tracheotomy, and in paralysis of the throat muscles as in diphtheria and poliomyelitis. The tube may be passed through the nostril and down the esophagus into the stomach with very little difficulty.

Organotherapy has gained for itself a well established place in pediatric therapeutics and probably has a very promising future. Those most used are extracts of thyroid, thymus, pituitary, adrenals, testes, and ovary. In the cretin the thyroid has brought about marvelous cures. Early in cases of mental impairment good results have been obtained from mixed glands. In slow development, also, mixed glands have been used advantageously. There has been, however, quite a tendency toward the use of mixed glands, and it would seem that some specificity should be established for best results.

In infancy and childhood nutrition is most important because upon the proper selection of food and regulation of habits depend growth and development. It is true that nutrition plays a very important role in adult life, but in infancy and childhood, where the body's chief concern is growth, nutrition has a peculiar significance. The food elements are the same and even the required percentages of these elements are the same as for adults. In fact, the percentage of fat, carbohydrate, and protein of mother's milk are the percentages upon which the adult body does best. On account of the growth of the infant or child the relative amount or value of food required greatly exceeds that required by the adult. For instance, in the young infant it is usually estimated that 100 calories per kg. or 45 calories per pound, and at one year 70 per kg. and 30 per pound, are required to meet body energy and growth. In the adult only 35 to 40 calories per kg. are necessary. As the infant grows the caloric requirements, or the amount of food per kg. of body-weight, lessens. At the twelfth year it is estimated that the food requirement is 25 per cent. greater than that of the normal full-grown adult.

Because of these facts it is readily seen why specific dietary is so essential in pediatrics. Caloric values are used only to check up the amount of food taken. As in adult life, not only is it essential that the different food elements are in the right proportions but food must be in certain definite forms, that the delicate structure of the infant's digestive system be able to assimilate it. Excesses of the different elements are not so readily taken care of as they are later in life.

In the infant's dietary special attention must always be given to supply the necessary vitamins. Many nutritional disorders are due to their absence. Scurvy is not necessarily a disease of infancy but is seen at any age where fresh foods are absent from the diet. Nevertheless it is seen much more frequently in infancy because of the especially prepared foods and the use of proprietary articles of diet placed before the public, most of which are without the necessary vitamins.

During the first 9 or 10 months the natural food for a baby is mother's milk. Unfortunately, many mothers do not supply their young with this food, and artificial feeding is necessary. That is, it is necessary to supply the infant with food from some other source. The milk from animals naturally has been first resorted to, and the one most depended upon is the cow's milk. This milk is modified by dilution with water and addition of various carbohydrates. At times it is necessary to change the proportion of one or other of the elements of the milk. Each infant's ability to use artificial feeding varies, consequently the need of individual feeding prescriptions.

The idiosyncrasies toward food, or the inability to digest one or the other of the food elements, must be taken into consideration and avoided. Many disturbances are due to a sensitization to one or more food proteins, which must be determined and that food eliminated from the diet. As the child grows it naturally requires an increased quantity and quality, but all the while under definite medical supervision, because a great percentage of digestive disturbances arise from too much food or from an unbalanced dietary.

The dietary of feedings must depend entirely upon individual body requirements, taking into consideration the temperament of the digestive cells.

Improper feeding, including improper quality, quantity, and constituents, cause the so-frequent derangement of the infant's digestion. Both stomach and intestines, undeveloped as they are, are quite easily disturbed and inflamed, and the acute dyspepsia and infections readily appear. The failure to correct the dietary errors leads to a subacute or chronic disorder and an inability to assimilate any food. It is then we see the infant suffering from inanition and marasmus (decomposition).

In older children dietary errors lead to indigestion, both gastric and intestinal, an absence of growth, and a failure of development.

In infancy and childhood the condition known as acidosis is frequently encountered, while in adult life it is rare. It is the using up of the alkali reserve, either by its over-elimination or by the over-production of acids. It is a carbohydrate starvation. Its prevention is dependent upon the preservation of the normal alkalinity in the body fluids, that is, the ingestion of proper foods and alkali, and the avoidance of conditions increasing body acidity. In starvation, in fright, in severe nervous strain, after operations, even in the common homesickness and after illnesses, the normal alkalinity is upset and acidosis, to a moderate or more severe degree, is present. Naturally, in the infant and child, when the digestive system is delicate, when the nervous sytem is unstable and delicately balanced, this condition would be much more frequently developed.

In infancy the peculiar constitution of the cell frequently portrays itself. The infant formerly classed as scrofulous, who shows excoriated lesions of the skin about the nose and mouth, or eczema of the skin of the scalp, face and body, with enlarged lymphatics, soft flabby subcutaneous tissues and muscles, rachitic bones, impaired digestion, and an ever-present respiratory tract disturbance, suffers the peculiar condition known as exudative diathesis. It probably is caused by a constitutional peculiarity in the cell, possibly a chemical change. With these children fat is usually ill-borne, but carbohydrate is well taken. From these peculiarities in the required diet we might infer that there exists some disturbance of the chemical reaction of the tissues, probably an acid predominating.

The neuropathic infant, who is the offspring of neurotic parents, is inheritant of a peculiar neurone and unfortunately is placed in neurotic associations. This child is restless, crying constantly, develops vomiting and diarrhea with any food, and convulsions with any irritation or fever. It seems that in the feeding of these infants sugars are frequently very poorly borne but starches well borne. Protein is also well taken. Placing these youngsters away from unfavorable surroundings seems of most benefit. The neuropathic child often is the subject of improper diet and care. Stimulants, such as coffee, tea, and excessive sweets, and irregular meals as well as late hours, are permitted by the parents.

Malformations of the various parts of the digestive system, of course, are peculiar to infancy. One of the most frequent congenital deformities is harelip, associated rather often with cleft-palate. Rarely hypertrophy of the tongue or lip is encountered. Much more frequently tongue-tie is seen, and the bifid uvula. Congenital constrictions or atresias of the esophagus and of any part of the bowel are not uncommon and even the imperforate anus occurs. An hypertrophied stenosis of the pylorus is not infrequent. This condition seems to be congenital and appears as a rule in the first-born of a nervous mother. It is more frequent in the male child. Fluid or foods taken are immediately and violently expulsed. Stools are scant or absent. As soon as food enters the stomach its outline becomes visible through the abdominal wall and waves of peristalsis are quite easily seen. Oftentimes a mass is felt in the pyloric region. Definite hypertrophic stenosis requires surgical interference. At times the pylorus and upper duodenum is a thickened cord with the lumen very small or none at all. The symptoms are the same as in hypertrophic stenosis but no mass is felt. There is, however, another condition which simulates stenosis. This is pyloric spasm. Here the expulsion of food is not constant, the gastric distention and peristalsis not so marked, bowel movements are larger showing that more food passes into the intestine. The child does not suffer the gradual wasting seen in the true type. Treatment by proper selection and regulation of food, possibly with medication to overcome hypersensitiveness and nervous excitement, is indicated and is usually successful.

Inflammatory conditions or diseases of the mouth are common in infancy. They are due to neglect and usually unhygienic surroundings. They vary from simple catarrhal type through the aphthous, ulcerative, parasitic, infectious, to the gangrenous. An inflammatory condition of the mouth is readily prevented if the child be given after feedings a small amount of water to remove remnants of food particles, and by the avoidance of dirty, infected articles with which the child comes in contact. Naturally the child puts everything in its mouth; therefore the necessity of a cleansing, mildly antiseptic mouth wash, harmless if swallowed. Then too, gastric disturbances may cause acid eructations and their consequent irritations. In the protracted exhausting diseases, infectious and parasitic types of stomatitis are frequently encountered and are to be avoided only by the most careful hygiene. In scurvy the oral mucosa is congested and easily infected. Gangrenous stomatitis, or noma, is rare, and usually secondary to one or other of the infectious diseases. It is not infrequently epidemic in institutions. It attacks those of poor general nutrition, developing from an ulcerative type of stomatitis. The lesion begins as a small spot, spreading rapidly to eat away a large part of the cheek and face. The odor of putrefaction is terrible. The disease can be prevented but cure seems impossible.

Dentition has given rise to much discussion as an etiological factor in disease. Too much has been blamed upon teething, but it is undoubtedly true that there is more or less irritation caused by the eruption of the teeth. This varies in different infants, in some causing quite marked disturbance while in others seemingly none at all. Undoubtedly most of the disturbances of the digestive and respiratory tracts at this time are due to the nervous agitation which arises. The skin lesions so often referred to as "tooth rash" are probably directly due to upset digestive and nervous function.

If in early life proper food is supplied, early use of the jaws encouraged to obtain proper mastication of food and development of jaws and teeth, and a reasonable amount of dental or oral hygiene insisted upon, much of the distressing condition of later childhood can be prevented.

The diseases of the digestive tract make up a great proportion of the diseases of infancy and early life. For the most part these are dependent upon improper feeding and infection. It must be remembered, however, that even at birth all the digestive juices are present and capable of functioning well so long as the food is given in proper amounts and in *suitable preparation.* Indiscretions in food of the infant and child are much more likely to disturb the entire organism—and more severely.

The stomach of the newborn baby is more vertical in position than in later life and for this reason vomiting and regurgitation of food is provoked with the slightest disturbance, even in manipulation or change of position. The cardiac orifice, too, is less efficient. The stomach also propels the food through the pylorus very early, and after the first five minutes of feeding, food is in the intestines. This is especially so in the breast fed babies.

Gastric digestion is less important in infancy, owing probably to the one type of food used. The coagulation of the protein is the only necessary preliminary preparation in the stomach. Artificial food is more slowly taken care of. The greater part of the digestion of the food takes place in the intestine. Absorption takes place very rapidly in the intestine after the foods are broken up. In the large intestine for the most part water is all that is absorbed, although there is a slight absorption of sugars, salts, and peptones. The small intestine is 9 ft. in length at birth, and the large intestine 18 in. long. The sigmoid flexure is very long and equals almost half the length of the large intestine.

The intestinal bacteria are absent at birth, but very shortly thereafter, or as soon as milk is ingested, the usual bacterial flora enters. It is the same as in the adult only when the child is given a mixed diet.

The normal bowel movements of breast fed babies differ from adult stools in that they number two to six a day. They are looser in consistency, lighter in color, with an acid or slightly sour odor. The infant fed on cow's milk has a more pasty appearing stool, pale yellow, reaction neutral or alkaline. The only gases present are hydrogen and carbon dioxide, and it is due to this absence of sulphur hydrogen and marsh gas that the odor differs from the adult stool.

Recurrent or cyclic vomiting is a condition not infrequently met in children, usually between the ages of two and four years, although the attacks may date back into infancy. It is characterized by periodic attacks of vomiting, which occur at rather regular intervals and apparently with no definite cause. The attack will last for two or three days and all attempts to control it seem fruitless. Thereupon the child assumes its usual state of health. These children are usually somewhat delicate and high-strung nervously. Probably the only exciting cause may be a nervous one such as a railroad journey, attendance at a show, or after a fatiguing interval of play. There are indeed the same etiological factors as producing acidosis, of which this is one type. The attack is preceded by a period of lassitude, languor, yawning, loss of appetite, heavy eyes, soon followed by sudden vomiting which persists. The slightest amount of food is ejected, and even when no food is taken vomitus consisting only of gastric juice and mucus, at times blood-streaked, is brought up. There is a slight elevation of temperature and the child becomes exhausted, sometimes to the point of threatening life. After 36 to 72 hours the attack ceases and the child convalesces rapidly. The attacks recur, becoming less frequent until the child is 10 or 12 years old. It must be remembered. however, that in these cases acetonuria may not be present until the last part of a severe attack, and even the foulness of the breath may disguise a fruity odor. Some constipation is usually present, but there may be diarrhea. There is little danger to life, although death has been recorded in quite a few instances. Starvation, alkaline enemata, and free purgation early in the attack are most serviceable, while during the attack fluids must be kept up and soda-bicarbonate or dextrose administered by enteroclysis, hypodermoclysis, or intra-peritoneal injection. When the vomiting has ceased judicious feeding of carbohydrates and of small quantities of milk is indicated.

Inflammatory diseases of the stomach are rare in infancy unless accompanied by inflammation of the intestines. The inflammatory diseases of the intestines are much more frequent. The stomach resists infection—the intestine does not.

Improper food and infection are the most important causes of any disturbance. The lesions are, as in adult life, catarrhal, ulcerative, and at times membranous.

In older children gastritis is seen more frequently, and here too indiscretions of food are the principal causes. There is frequently alarming high temperature or convulsions. Treatment does not vary from that used in later life.

Distention of the stomach is seen not uncommonly in overfed infants, in infants suffering with rickets, and in those cases where there is some obstruction of the pylorus. In older children the distention usually accompanies some chronic indigestion (fermentation) and muscular atony. The diagnosis is made by the outline of the stomach as shown through the abdominal wall, and by percussion.

Ulcers of the stomach are not nearly so frequent as ulcers in the duodenum. Areas are oftentimes found post-mortem, but unless the examination has been made immediately following death it is impossible to say that they existed during life. Duodenal ulcer is found frequently in cases of severe malnutrition, and in toxic cases such as severe cutaneous burns. These ulcerations are probably due to toxic material from the liver. They are diagnosed only by the finding of occult blood in the stools and in the vomitus. Treatment is symptomatic and the avoidance of continued irritation. At times surgical interference may be necessary. At times a localized peritonitis may be found post-mortem, indicating such a lesion perforated.

Hemorrhages from the stomach are seen in the melena of the newborn, in purpura, hemophilias, scurvy in older children, and at times in young girls at the age of puberty as vicarious menstruation. In cases of severe vomiting hemorrhage is very likely to occur, or where there has been any irritation, either chemical or mechanical, such as a foreign body.

Diarrheal diseases are the most frequent form of disturbance in infancy, and the infant mortality is greater from diarrheal diseases than from all the acute infectious diseases combined. The frequent loose evacuation of the bowels results from improper food, infection, unhygienic surroundings, and neglect or ignorance of the mother. They are by far most prevalent during the summer months because of the greater likelihood of food infection at this time. How atmospheric heat acts in causing diarrheal diseases is not yet entirely settled, but it exerts possibly some slight depressing effect on the infant, especially the extreme hot weather. The greatest numbers of these cases are seen in the cities. Baby welfare work and the establishment of milk stations and pure milk supply has done much to decrease the number of such cases. Encouragement of maternal nursing, education of the mother in the care and hygiene of the child, supervision of the milk supply, and instruction and supervision in cases where artificial feeding is necessary have lowered the infant mortality markedly.

The definite diagnosis of intestinal disorders depends upon the thorough examination of the stools and a complete history of feeding, even previous to the apparent onset.

Chronic intestinal indigestion is seen commonly in children under five years of age, probably accompanying a general weakness; or it may be the result of long continued improper feeding. It is most usual that the diet is at fault, and too great an amount of food is taken or that the food elements are out of proper balance. Improperly prepared food is an etiological factor as well as improper articles of food, such as coffee, sweets, etc.

These children are usually thin, underweight, delicate, with a large and distended abdomen. They are consequently subject to all the acute disorders. Diagnosis is made by careful inquiry into the diet, and examination of the stools.

Colic of infancy is caused by a spasm of intestinal musculature. It is a reflex irritation, caused by the gas of fermentation of undigested food, due to improper feeding. Colicky pains are seen in all forms of intestinal inflammation, in intussusception, and in appendicitis.

Constipation in infancy is usually due to improper balance of food in the artificially fed, and to constipation of the mother in the breast fed. A fissure in ano is a very frequent cause. It may also be caused by the habitual use of suppositories or laxatives. Far too frequently the infant is given castor oil, milk of magnesia, cascara and so on. Temporary relief is obtained—the underlying cause is not removed, and constipation is more persistent. An atonic bowel musculature may be its true cause, or possibly too small an intake of water.

The condition known as Hirschsprung's disease is met with in early life. The abdomen is greatly enlarged, due to a greatly distended colon which is probably the result of some low obstruction or to an overly elongated sigmoid. The "Megacolon" has been attributed to an intra-uterine inflammatory disease. It is distended with gas and fecal matter and its walls hypertrophied. It is quite readily outlined by percussion. The condition may appear early—that is, during the first year; or it may appear by slow development up to the third or fourth year. The bowel movements are usually dry hard masses, and occasionally a diarrheal stool may appear. At times these children suffer with attacks of vomiting or slight persistent indigestion. The child may appear normal for quite a while, and then gradually, as the condition becomes worse, the general nutrition fails. Treatment consists of careful feeding and special attention to the evacuation of the bowel. In severe cases surgical interference will probably be necessary.

Intussusception is by far the most frequent form of acute intestinal obstruction in infancy. It is not at all uncommon and prompt recognition and operation are essential to life. Usually the ileocecal part of the intestine is involved. Three-fourths of the cases occur in the first two years of life, 50 per cent. between the fourth and ninth months. Males seem more frequently affected than females, and most frequently the patient is one in apparent "super" health. Probably there is always some preceding intestinal disorder, but it may be very slight. In a minority of cases there is a definite diarrhea or constipation. The frequency of intussusception in infancy is probably due to the very thin walls of the intestines, and to the free mobility of the cecum and colon. The chief symptoms are sudden in onset, with severe pain and vomiting. The cry recurs in paroxysms. The vomiting consists first of gastric contents, and then of bile with a fecal odor. There may be one or two bowel movements with fecal material, and then only blood or blood and mucus. The child is severely prostrated, the abdomen is tense, and the thighs flexed upon it. The first temperature may be normal or subnormal, but later it rises constantly so long as the condition exists. There is always a sausage-like tumor mass palpated through the abdominal wall and a definite mass felt per rectum. Diagnosis of the condition is quite readily made. The presence of the characteristic tumor is conclusive. The treatment is surgical. The prognosis depends upon the age of the patient and upon early recognition and operation. After the second day the chances of recovery are almost nil. The general mortality is 50 per cent. There is some probability of a spontaneous reduction in some few cases of intussusception but this is a matter of doubt in the minds of a great many pediatricians.

Appendicitis is met with at all ages, but it is rare in infancy. In older children it is not infrequent, the largest number occurring between the ages of 10 and 15 years. In the older children the attack is the same as in adults and the same treatment is indicated. It must, however, be remembered that in children appendicitis in a very short time, even four to six hours, is likely to become a pus appendix. The condition is met with occasionally in infants and diagnosis is made by the elimination of other frequent abdominal conditions. There is fever, the distinct localized rigidity, sudden onset, pain, and increased polynuclear leukocytosis. Tenderness is more often higher or to the back. Rectal examination is especially valuable. In infancy localized peritonitis is of rapid development. The younger the child the less favorable the prognosis. Special differential diagnosis must always be made in the young between psoas abscess, intussusception, and diaphragmatic pleurisy at the onset of pneumonia. Diagnosis frequently is not made until the appendix is ruptured. Fortunately infants do well after drainage.

Intestinal parasites are not uncommon in children but they are far more infrequent than is usually supposed, especially by the laity. The most frequent worms infecting the bowels of children are thread worms, round worms, and tape worms, in the order named. Diagnosis is only made by the finding of the worm or its ova in the feces. A high eosinophile presence in differential blood suggests the possibility of parasites. The usual rubbing or picking of the nose so frequently referred to as a symptom of "worms" is more likely only a symptom of nasal disorder. Treatment depends upon the determination of the parasite present.

Prolapsus anæ is frequently seen in the marasmic baby suffering from diarrheal disturbance or anything which provokes severe prolonged straining. It is most commonly seen in the second year of life, usually then from the straining of severe chronic constipation. Bright red blood is usually on the fecal masses, and the prolapse tumor is visible. Reduction is accomplished by gentle pressure and cold applications.

Fissures of the anus are quite common in children and especially in those suffering from constipation. In infancy it is not at all infrequent and is from injury by hard fecal masses or from suppositories or syringe nozzles. Irritation by pin worms may cause a fissure or an inflammation of the rectum. Infection about the rectum is frequently the cause of ischiorectal abscess, a less likely cause being traumatism. Inguinal adenitis is usually complicating.

Acute peritonitis may be seen in the newborn originating from an infected umbilicus. In later childhood it is occasionally seen secondary to other conditions, the same as in adults, especially appendicitis. As a primary inflammation it is rare. Tuberculous peritonitis is sometimes observed between the first and fifth years. The mesenteric glands are first involved, these breaking down and general involvement of the peritoneum following. It may be either fibrous or ascitic. The former exists more commonly. It is slow in development and produces extensive adhesions between the serous coats of the intestines and the abdominal wall. Naturally the serous membranes are greatly thickened as well as the omentum and fluid may accumulate, serous or purulent. The general health of the child remains good for a time; there is no fever; the bowels may be constipated or loose; but all the while there is a distinct enlargement of the abdomen. This enlargement is first due to tympanites, and later to the thickened serous membrane, lymph-nodes, and fluid. The fluid may vary—be present for awhile and then disappear. Gradually there is an increase of intra-abdominal pressure symptoms, indigestion, emaciation, and wasting. Fever gradually appears, ranging from 99 to 102 degrees and higher. The child becomes anemic and distinct doughy masses or thickenings can be palpated through the abdominal wall. Pressure upon the pleura causes congestion and pulmonary involvement from which as a rule death results. The duration usually is three to six months. Death may be from disseminated tuberculosis, tuberculous meningitis, or general exhaustion. As a rule, wherever there is chronic enlargement of the abdomen with ascites and fever, a diagnosis of tuberculous peritonitis may be made. Tuberculous peritonitis is always a serious disease but there have been some recoveries. The younger the child the more grave the prognosis. Those cases with ascites are more hopeful. The general treatment is the same as for tuberculosis elsewhere.

The liver in the fetus belongs to the hemopatic system, although in a very minor way it does produce bile, store glycogen and detoxify. At birth and during infancy it is proportionately twice the size of the adult organ.

Distinct disease of the liver is infrequent at this early age, although its involvement complicating all other disorders of the body must be of utmost significance if one recalls that it is the first organ to receive the intestinal substances assigned for digestion, and the organ which receives all toxic or infectious substances from any source whatsoever, The nature, degree, and effect of these conditions are indefinitely recognized and little understood.

Jaundice is the most constant symptom of disease of the liver. Its appearance in infancy suggests the frequent icterus neonatorum, an umbilical infection, a septicemia, a congenital family cholemia or possibly a tumor growth. It is present too in the rare diseases of the liver described by Babel and by Winckel.

In older children jaundice coming on acutely is catarrhal, while a persistent or recurrent one would suspect a cirrhosis, a yellow atrophy, a congenital family cholemia, or more rarely gall-stones or hydatid cysts. An abdominal tumor might cause sufficient pressure.

In early life the abdomen is frequently enlarged. It is most commonly due to a fatty infiltration as found in metabolic disturbances accompanying syphilis, tuberculosis, rickets, and chronic indigestion. The less frequent fatty degeneration is found in intoxications.

Pancreatic disease in infancy and childhood is mostly functional. Lipase, protease, and amylase inactivities are often encountered, the disturbance varying in intensity in the order named.

Celiac disease, pancreatic or intestinal infantalism, is a type of chronic intestinal indigestion and concomitant delayed growth. Acute inflammatory disease of the pancreas occasionally accompanies the acute infections, especially mumps. High abdominal pain and tenderness, shock, diarrhea and vomiting may be quite sudden and alarming. The condition usually clears up in a day or so with symptomatic treatment.

Diseases of the respiratory tract are second in frequency to those of the gastro-intestinal tract. There is a definite familial predisposition to inflammatory diseases of the respiratory mucous membranes. It is seen in those who have inherited malformations of the nose and extensive presence of lymphoid tissue. An overgrowth of the adenoid and tonsillar tissue may, however, follow an infection which is acquired from parents or associates who are frequent sufferers from nasal "colds." More frequent involvements of the respiratory tract are observed in those children who live in unhygienic surroundings, and in those improperly fed. High carbohydrate feeding is conducive to infection of the mucous membranes. In infants, too, it must always be remembered the nasal passages are smaller than in the adult, and consequently a slight congestion occludes them. A slightly deviated septum is more obstructive in the child than in the adult. A slight infection of the nasal mucous membranes almost invariably involves the naso-pharynx and may extend quite rapidly in the young child to the larynx and bronchi. The Eustachian tubes, being shorter in length and larger in caliber, frequently become involved. The treatment varies little from that of adults. Most important is prevention. Obstructions and focal infections should be removed, the surroundings should be hygienic, and plenty of fresh air should be afforded. Proper dietary should be established and association with persons who have acute or chronic respiratory disorders avoided. In chronic "snuffles" one should always consider syphilis, rickets, and nasal diphtheria.

Acute tonsillitis is common in infancy and is frequently overlooked because of an apparent absence of pain and difficulty in swallowing. It is the precursor of cervical adenitis, nephritis, endocarditis, and chorea, and only by careful examination of the throat in each illness can mistaken diagnoses and embarrassing sequelæ be prevented. In any membranous involvement diphtheria antitoxin will do no harm and should be administered if the slightest doubt of diagnosis exists. If one waits for a cultural report serious progress of the disease may have been made.

Peritonsillar abscesses are not so frequent in infancy, but the retropharyngeal abscesses are more frequent than in adult life. Dyspnea and suffocating attacks are the prominent symptoms.

Involvement of the larynx is much more frequent in infancy and childhood. It is always more or less associated with muscle spasm, resulting in the hoarse, tight, and then brassy, croupy cough. Croup, or catarrhal inflammation of the larynx, is peculiar to children, especially from the sixth month to the fifth year, when the recurrent attacks seem to disappear. It is more often seen in the baby with an over-abundance of fat and one who exhibits overgrowth of lymphatic tissues. These youngsters are the ones who are especially active and who readily become over-heated. They are usually the ones who over-eat. The attack may appear suddenly at night after a day of over-exercise or excitement or after being out in a high wind. It may follow indiscretion in eating and appear with an indigestion. It is treated by emetics, inhalations, hot draughts, and hot or cold local applications. Usually it is well to employ sedatives such as bromids or small, carefully estimated doses of opiates. It is peculiar, too, that the attack is likely to recur during the next two or three nights, the child during the daytime showing no other discomfort than that from a nasal "cold."

Edema of the glottis is rare in infancy. In laryngeal disturbances in childhood it is most essential that one keep in mind the possibility of laryngeal diphtheria. This is always slower of onset than croup, and the symptoms gradually increase. The child has been ill for a day or two, and has had fever. There has been a nasal discharge, possibly a complaint of sore throat, possibly a complaint of pain in back and legs. The respirations are labored. With inspiration there is an indrawing of the intercostal spaces and the muscles of respiration are over-active. There is the dry cough, hoarseness, and croup. In all cases of laryngitis a most careful examination of the throat and nose should be made. A membrane or a bloody discharge is sufficient for serious consideration. In most cases of diphtheria there is a characteristic odor which is of most valuable diagnostic importance. In any case where there is the slightest doubt smears and cultures should be made. Antitoxin is of course the only treatment. Preparation for possible intubation or tracheotomy should always be considered.

Foreign bodies are oftentimes drawn into the larynx and bronchi by children. Diagnosis might possibly be made by the laryngoscope, but it is frequently necessary to employ the fluoroscope or *x*-ray. Removal may sometimes be effected by a violent cough, but frequently only through a bronchoscope.

A dry, harrassing and persistent cough suggests some pressure upon, or irritation of, the trachea, bronchi, or nerve supply. It may be, too, a reflex irritation, as in an otitis media. An enlarged or persistent thymus, or enlargement of the tracheo-bronchial lymph-nodes, may cause pressure or irritation on the trachea or its bifurcations, or upon the recurrent laryngeal nerve. Diagnosis is made by *x*-ray examination. The tracheo-bronchial lymph-node enlargements are usually secondary to cervical node involvement and frequently tuberculous. Other causes might be a sarcomatous growth or a mediastinal disturbance. In tuberculosis it is not infrequent that a lymph-node may rupture into one of the bronchi. This rupture usually takes place with a violent attack of coughing, suffocation, and then an expulsion of a little blood and caseous pus.

The respiratory organs in infancy and early childhood exhibit several peculiarities which must be carefully kept in mind in studying their disease. The trachea, bronchi, and bronchioles are larger in caliber than in the adult. The lung tissue, therefore, is less in amount; the chest wall is thinner, giving a peculiar puerile type of respiration which is many times mistaken for pathological. The air vesicles or sacs are smaller, the walls very delicate, so that any inflammation of the larger bronchi may extend rapidly and involve them. Their congestion is frequent and consequently they are affected almost as much as if they were consolidated. The tender lining of the air vessels is much more subject to collapse as well as to rupture and to emphysema. These two conditions are, however, only temporary, recovery taking place as a rule. Consolidated areas may readily exist in the lung of the infant and show no impairment of percussion note. Often even the auscultatory symptoms are hidden. About the consolidated area the lung is emphysematous or healthy, and this obscures the findings which one would naturally expect. Bronchial râles are commonly heard and misinterpreted, as well as the exaggerated puerile breathing which is confounded with the tubular type. Fluid may exist in the chest, giving a flat note, but breath sounds may be bronchial.

Acute catarrhal bronchitis is a most common ailment in children of all ages. The acute rhinitis and coryza extend rapidly into resultant bronchial inflammation. In all infections bronchitis co-exists, and the predisposition to bronchitis exists in all nutritional and debilitating diseases. It is usually secondary to disease higher up in the respiratory tract and its etiology and treatment are the same.

Children who suffer chronic debilitating diseases (as tuberculosis, syphilis or, in older children, heart disease) or from frequent bronchial inflammations are subject to chronic bronchitis. It is not frequent. The treatment of this condition is not unlike that of adults—removal of cause and change of climate.

Infants and children suffer from asthmatic attacks as do adults. The infant or young child shows with each bronchial disturbance a symptomatic asthma. It is very frequent in children to find asthma with its characteristic urticaria and respiratory symptoms. Pollens, contact with animal or food proteins are causes, and the susceptibility is frequently handed down from parent to child. Emphysema is a rare condition in early life.

Pneumonia is a common disturbance in infancy and childhood. It is one of the largest factors in infant mortality. Bronchopneumonia is usually secondary to other diseases or an extension of higher respiratory involvement. It is on this account probably the most common form of pneumonia seen in infants in the first and second year. It is doubtfully ever primary.

Lobar pneumonia is seen more frequently after the second year, but it is not at all uncommon before that time. It has a much more favorable prognosis both in infancy and childhood than in adult life.

The younger the infant, however, the more dangerous the disease. It is true that lobar pneumonia usually affects the child who was previously healthy and bronchopneumonia the run down child. There is no condition in which the child seems so severely "hit" as in lobar pneumonia and makes complete recovery. It is often accompanied by severe meningismus which is most alarming. The onset with violent vomiting or abdominal pain is quite confusing. The area of consolidation is often first the hyper-resonant area and oftentimes the true physical findings may not be demonstrated until the disease is subsiding. It is because of these delayed physical signs that lobar pneumonia has been popularly supposed to be less frequent than bronchopneumonia. Positive blood cultures are far less frequent in children than in adults. Treatment varies little from that of adults. Rest, relief from fatigue caused by coughing, fresh air, and good nursing are essential.

Empyema is a frequent sequela of lobar pneumonia in children and the prognosis is favorable (especially after infancy) with early recognition and treatment. Purulent pleurisy is the most frequent form with which the pediatrician deals—dry pleurisy is less frequently encountered. Serous pleural effusion may follow a bronchopneumonia and rarely a purulent one is found. This is frequently the case in the influenzal infections.

Atelectasis is not an infrequent congenital disturbance. As a rule it is seen in the premature or congenitally weak. A prolonged hard labor may be a sufficient cause. It is never to be feared or anticipated when the child cries lustily. The acquired form is usually due to some aspirated obstruction.

A pneumothorax is almost unheard of in early life. Some air, however, will always be found co-existing with a pyothorax.

Acute cardiac disease is almost never found in infancy—in fact, one very rarely finds an acute cardiac lesion until after the third or fourth year. If there is an acute lesion, the mitral valve is the one usually involved; but to make such a diagnosis one must have a definite history of some preceding infection. This may have been pneumonia or tonsillitis; it may have been a furunculosis or scarlet fever, or some focal infection. A systolic murmur at the apex, transmitted into the axilla or back, is alone not sufficient for diagnosis. It must be accompanied by cardiac enlargement and decompensation. Anemic, functional, or possibly a slight congenital defect, may give the murmur and its transmission. After the third or fourth year one very frequently finds evidence of cardiac disease with signs and symptoms almost the same as those found in adult life. Endocarditis is very likely also a myocarditis, and a very rapid pulse is counted. Pericarditis is also occasionally found and it must be remembered that in infancy and early life pus formation is much more rapid than in later life. The prognosis of cardiac disease in childhood is quite good if the original infection can be located and eliminated. Recurrent attacks of rheumatism, tonsillitis, and cardiac involvement of course make the prognosis more grave. In the treatment of cardiac disease in children absolute rest is most important, as well as prolonged rest, with thereafter most careful observation and protection. Nephritis affects the prognosis very unfavorably. It is, however, very frequently observed that a child who has suffered an acute carditis with all its signs and symptoms is found later to possess a perfectly good functioning heart, even to the extent of no enlargement and no murmurs. Some clinicians doubt that this was really a valvular involvement and suspect that the whole condition was possibly only myocardial. Whether or not this is true depends upon very careful pathological findings in recorded cases. Cardiac disease in later life probably had its incipiency in neglected youth, hence the prevention of cardiac disease in infancy and childhood is of imperative importance. This means the prevention, the proper care, and the elimination of infections.

The signs and symptoms of congenital heart disease appear at birth or shortly thereafter. The child may be seized with sudden attacks of dyspnea, cyanosis, or pallor. There is a loud, harsh murmur heard over the precordium and even at times throughout the chest. After a few years the precordium is usually bulging and the child suffers not only from cardiac embarrassment but also from a general body malnutrition. Most of these babies with a congenital heart involvement live only a few years, some intercurrent infection or disease being the final illness. However, those with minor lesions often live well into adult life and with very little embarrassment. Definite diagnosis of the lesion is at times quite difficult and one oftentimes must be content with the diagnosis of a congenital heart.

Arterial disease is never found in infants. Angiomata or nevi are of course congenital and they are extremely frequent, although as a rule they are the very mild cutaneous birth marks. The more extreme involvements are rare and only in the extreme types is there any danger to life.

The newborn blood normally has a very high hemoglobin and cell count, but after 10 days or two weeks the normal is reached, except that the white cells continue at from 10,000 to 12,000 into childhood. This is a lymphocytic increase. Aside from the polymorphonuclear leucocytosis in sepsis, the lymphocytosis in pertussis, the eosinophilia in intestinal parasites, and the possible leucopenia in malaria, typhoid, and influenza, the number and form of white cells suffer very few variations in infancy.

Anemia is much more readily produced in infancy than in later life and it is just as much more easily overcome. Secondary anemia is very frequently found in early life and it might seem that childhood is disposed toward it. Growth taxes the blood to a great capacity and any slight disordered health overtaxes it. The newly born is anemic if the mother be in poor health or if she has suffered from any acute or chronic disease during the pregnancy. The hemorrhages to which the newly born are subject produce this disease. As the infant grows, any infection, any respiratory disturbance, and especially any gastro-intestinal disturbance, impoverishes the blood. Secondary anemia is found in rickets and in those babies whose surroundings and food are unfavorable. It is often overlooked as a cause for poor nutrition, and very frequently overlooked about the age of puberty.

Chlorosis is a peculiar blood disease seen in girls at the age of puberty. It is not seen in earlier life. The patient has a peculiar greenish-yellow tint to the skin but does not seem to be emaciated. There is a marked reduction in the hemoglobin but the red cells are not reduced in proportion. It rarely occurs in males, but more especially in the light complexioned females. The exact etiology is unknown but it is seen more frequently in those who suffer from poor care, poor food, and poor ventilation. The patient with chlorosis suffers shortness of breath, dizziness, palpitation, fainting attacks, indigestion, constipation, and a failure to establish menses. The slightest exertion overtaxes her, and with all this the appetite is enormous and she craves all sorts of food, mostly the indigestible. Anemic murmurs are always heard over the heart and a venous hum throughout the vessels. The heart may be slightly dilated and the pulse weak and soft. The urine may show some albumin and the feet some puffiness. There is general malaise, and indefinite aches and various neurotic and hysterical manifestations. There is hyperchlorydria and ulceration of the stomach is not infrequent. The disease is cured slowly by the improvement of the manner of living and supplying of wholesome food.

Infants at times show a form of anemia which by some is called a pseudoleukemic anemia (so-called "splenic anemia" and "von Jaksch's anemia"). It is characterized by high leucocytosis, a reduction in red cells and in the hemoglobin, a very large spleen, and at times an enlarged liver and lymph-nodes. It occurs mostly between the ages of 7 and 12 months and its cause is unknown. Its chief characteristic is the large spleen—firm, hard, and may show some evidences of inflammation of its capsule. The tissue is simply hyperplastic. The enlargement of the liver and lymph-nodes is less constant than that of the spleen and the tissues are unchanged. The hemoglobin may be as low as 25 per cent. and the red cells 2,000,000, with inequality in size and shape. White cells are increased and run from 20,000 to 50,000. Either the mononuclears or polynuclears may show an increase in the differential and all of the cells show degeneration. The disease is essentially a chronic one and the child is very pale. The prognosis should be guarded although most infants recover after several months.

Pernicious anemia is rarely seen in early life.

Leukemia is a rare disease of childhood but is seen at times, even in infancy. It often seems to follow other diseases.

Acute lymphatic leukemia is the most common form in early life and comes on as would an acute infection—abruptly, with fever, general pain, and marked prostration. Again, it may be gradual in onset with only the symptoms of swollen lymphatics which slowly increase. At times there seems to be bleeding into the subcutaneous tissues and the mucous membranes of the mouth may even be suggestive of scorbutus. The cervical and submaxillary lymphatics are markedly involved and the condition severe, with some elevation of temperature, dyspnea, and general weakness. The child wastes rapidly and the heart action is very weak and fast. The constant feature of the blood picture is the relative increase of the lymphocytes which may be as much as 90 per cent. of the whole white count, and a corresponding reduction in the polynuclears. The white cells may be from 20,000 to 150,000, especially later in the disease, and the red cells may be 1,000,000 to 3,000,000 with hemoglobin 25 to 30 per cent. The course of the disease is rapid, especially in the severe form, and death usually results in from one to three months from hemorrhage, exhaustion, or some intercurrent infection. There are milder cases which are attended by irregular and prolonged attacks of fever with varying symptoms. The lymphatic enlargement is constant. All forms of leukemia in early life have an unfavorable prognosis. Treatment is of little avail.

An atypical form of leukemia is chronic. The white count may be low but there are many myelocytes, and an increase of lymphocytes. The spleen, liver, and lymph-nodes are enlarged. Treatment is of little value.

Hemophilia is a disease suffered by the male children but transmitted through the females. These sufferers show a tendency to bleeding from the slightest wound and the hemorrhage is controlled with the greatest difficulty—frequently it is impossible to control it. The mother shows no signs of the disease herself, nor do her daughters. The blood shows an over-abundance of anti-thrombin. The disease is often discovered only by accident, and frequently too late. In some cases it seems that the disease might be outgrown, but it is the rule that one-half those so affected die before the seventh year.

Secondary purpura is seen frequently in children of all ages, even to the newly born, where it is spoken of frequently as Werlhof's disease. The infectious type is seen in the various forms of the acute infectious diseases such as measles, scarlet fever, variola, vaccinia, and typhus. Also seen in epidemic meningitis and at times in diphtheria and septicemia. The hemorrhages result from the altered condition of the blood-vessels. The prognosis of the disease from which it occurs is made much more serious by its presence. Purpura also occurs in cases of protracted illness. It is frequently seen in chronic gastro-intestinal diseases of infancy. The hemorrhages are into the skin and vary from petechia to large blotches. It usually appears on the abdomen and lower extremities and is commonly indicative of a fatal outcome. At times there are hemorrhages into the skin after the use of certain drugs such as quinin, phosphorus, and chlorate of potash. Frequently in the course of whooping cough hemorrhage may appear in the mucous membranes. It may also occur in epilepsy. After a protracted illness it may be seen in the skin, especially in the lower extremities, or it may be seen in surgical cases where splints have been used. The nervous or neurotic form of hemorrhage is seen infrequently in early life.

Primary purpura is frequently seen in children and appears in those who are suffering with poor nutrition, rickets, and anemia. It may, however, appear quite suddenly in those who are healthy. The hemorrhages may occur in any part of the body surface and vary in size. Gradually the infiltrated blood undergoes absorption. Both the skin and mucous surfaces are involved. At times there may even be bleeding from the bowel or kidney tract. Hemorrhage may occur with abdominal symptoms suggesting a surgical abdomen. It may occur with joint symptoms and suggest rheumatic arthritis. The pathology is imperfectly understood, and has been variously ascribed to changes in the blood-vessel walls, in the blood itself, and to the vasomotor control. There is very good reason, however, to attribute the disturbance to an infection.

The lymphatic tissues in early life are peculiarly prone to enlargement. In certain children this is much more marked than in others, and even in certain families there is more lymphatic hyperplasia. In any inflammation of the mucous membranes there is marked enlargement of the lymphatics which drain it. This is more marked in children than in adults and any inflammation of the lymph-nodes is more likely to suppurate. This is seen especially in the cervical adenitis following diseases of the nose and throat, and especially in the acute infections. In any case of adenitis in which the cause has not been removed the condition goes on to a sub-acute or chronic one and frequently this enlargement persists quite a period of time.

Hodgkin's disease, though rare, appears in childhood after the age of three years. It is seen more frequently in the older child. As in adults, the chief lesion is in the lymph-nodes. They become greatly enlarged and new nodes are involved as the disease progresses, both the external and internal, especially the mediastinum and the retro peritoneal. The first evidence is usually in one or more of the cervical nodes and the involvement progresses node by node until finally the general health is greatly impaired by malnutrition, anemia, and pressure. There is usually fever. The blood picture is the same as that of adult life and the duration of the illness is usually under three years—shorter than in the adult. Diagnosis is definitely made from a pathological section.

Splenic enlargement is more frequently found in children than in adults and is seen especially in rickets, syphilis, malaria, tuberculosis, and the blood diseases. Splenic anemia, or Banti's disease, usually has its onset late in childhood. The child at first suffers from anemia, with its dyspnea and palpitation, and then attacks of fever. The spleen is found moderately enlarged and firm, progressing slowly but never to a great size. Shortly thereafter there is noticeable enlargement of the liver, the health becomes undermined, and ascites occurs.

The liver then has diminished in size and the patient may show jaundice and dilated venules on the abdominal wall. Death usually occurs from some intercurrent disease. There is always some question as to the true identity of Banti's disease. Many conditions so diagnosed have proven to be syphilitic, and a great many, if not all, some authorities contend, are only cirrhoses of the liver. Others have possibly been Gaucher's disease.

Chronic family jaundice is occasionally seen and exists in several brothers and sisters while the parents may be unaffected. It seems to be transmitted through both sides of the family and not necessarily to all the children. It also seems that those children who are not affected do not transmit it to their offspring. Jaundice may be noticed very shortly after birth or may develop any time during childhood. It is the most striking feature of the disease and varies from a slight to a moderate discoloration. With any illness, especially with a disorder of the alimentary tract, the discoloration may temporarily become intense. There is always some anemia accompanying, both the red cells and the hemoglobin being reduced. The red cells show marked fragility, especially to hypotonic salt solutions. This condition does not disturb the growth and development of the child, but where there is an impairment in the child's general health surgical measures have been beneficial.

Gaucher's disease, though rare, attacks two or more members of one family, but does not seem to be transmitted through the family. Its onset is about the tenth year. The most characteristic feature is enlargement of the spleen which progresses until it may fill the abdomen. The mass is firm, smooth, and not tender. The liver is slightly enlarged. The skin has a peculiar brown discoloration, especially of the face, and at times the conjunctiva shows a yellow area. The general health is not disturbed for several years, the only symptom being discomfort from the enlarged spleen. Very rarely is there any jaundice or ascites. The true origin of the disease is not known but the pathological findings are distinctive and show that the disease is a systemic one. Death usually occurs from some intercurrent disease.

Cretinism is a fairly rare condition and depends on the absence of internal secretion of the thyroid gland. In many cases where autopsies have been performed the thyroid has been found to be entirely absent. The symptoms of cretinism usually appear during the second six months of life and even then are so slight that they might be disregarded until the child is two or three years of age. The first thing noticed is that the child does not gain in weight, mentality seems to lag, and then comes the characteristic facies. The head is large in proportion to the body. The anterior fontanelle remains open up until middle childhood. The forehead is low, the cranium seems thickened, the eyes set wide apart, nose flat, lips thick, mouth open and tongue protrudes. The eyelids are heavy and the hair of the head is coarse, straight and wiry. It often seems to be continuous into the eye-brows. Dentition is late and the teeth decay early. The body is usually very much dwarfed—the extremities heavy and very short. Bony growth seems to be absent. The skin is dry, cold, and harsh. The subcutaneous tissue is boggy. Umbilical hernia almost always exists, the abdomen is large, and all movements of the body are slow. Everything indicates mental and physical torpor. The body temperature is usually subnormal. Mentality is greatly impaired. The cretin has a hoarse, rough voice, is never excitable; speech is slow and often absent. But with it all the cretin is constantly good natured. If the condition is untreated there is failure of sexual development. The cretin may live to advanced age, always a dwarf, seldom attaining a height of more than four feet, with mentality never over the sixth year. Treatment of these cases gives remarkable results. Both physical and mental conditions approach that of the normal, but only if treatment is begun early.

There is another condition peculiar to children which is deficiency of the thyroid secretion, but in which the symptoms do not appear until the third to fifth year. The child shows moderate myxedema. These cases respond promptly to thyroid treatment.

Exophthalmic goiter appears occasionally in childhood, especially in females about the age of puberty. It differs from the disease in adults in that it is quick to develop and to disappear. It lasts as a rule only a few days and the prognosis is very good.

Frequently in females about the age of puberty there is marked restlessness, irritability, and nervousness. The thyroid is slightly and uniformly enlarged and other symptoms of hyperthyroidism are present to a moderate degree. Rest and establishment of menstruation usually overcome the disturbance. Whether or not these persons later suffer true goiter is a question to be determined.

Status lymphaticus is a definite pathological condition of which the etiology and pathogenesis is unknown. The striking characteristic of the condition is the enlargement of the thymus with hyperplasia of the lymphoid tissue throughout the body. Convulsions and attacks of asphyxia or of cyanosis or pallor occur frequently. Rickets is usually present, though not necessarily. Frequently the condition is not recognized until death with no apparent cause has occurred, and then only after autopsy. Diagnosis of the condition in life is often made by physical findings; a dull area is outlined by percussion at the base of the sternum. *X*-ray and fluoroscope are valuable aids. *X*-ray treatment seems to give good results.

Cases of infantilism are occasionally seen in children and are due to lesion (tumor) of the pituitary gland. The child is extremely fat and is mentally backward. There is an increased sugar tolerance and the sexual organs are infantile. Early endocrine treatment may sometimes be found beneficial.

Pituitary disease may also show evidence of a bony gigantism. This may appear in early years of life. Acromegaly does not appear until later life.

Disease of the parathyroids is a disturbance of the calcium metabolism, but the exact nature of the condition is not understood.

There is a great deal of endocrine therapy in practice at present, and as a matter of fact a great deal more than present knowledge would justify. However, the field for this division of treatment is of greatest possibilities in the fetus and in the infant. As our knowledge grows, possibly we will be able to understand and treat many of the conditions which at present are regarded as hopeless.

The urine in children differs in many respects from that in adult life. During the first year the excretion of urine per pound of body-weight is much greater than in later life. During the first few days it is very pale and of low specific gravity. It contains some albumin and uric acid crystals normally. Toward the end of the second year the excretion becomes less and is of higher specific gravity. Blood in the urine may occur in early life and be far less cause for alarm than later. It may appear with the passage of uric acid infarcts. During infancy it may be seen in melena or scurvy, or scrofulous disease of the kidney; in later life it may be seen in renal calculus or acute nephritis. At times there seems to be blood in the urine with no definite cause, especially in the latter half of childhood. It seems to come and go, quite suddenly, with no symptom of illness of any kind.

Suppression of urine is a very frequent symptom in infancy and the only definite cause seems to be from uric acid excess. When urination takes place, it is generally accompanied with pain and discomfort and examination discloses highly concentrated, uric-acid-filled urine. Local hot applications and water by mouth, with some alkaline, corrects the disturbance. In older children, painful urination is frequent, the result of errors in diet and some general physical impairment or lowered vitality.

Diabetes insipidus is probably found more frequently in childhood than in adult life. It seems to be sequent to some infection, as measles or influenza, at times syphilis, or at other times injury to the head. The child has an unquenchable thirst and passes enormous quantities of urine of low specific gravity, pale color, no sugar, and possibly a slight trace of albumin. Eventually, the patient becomes emaciated and general malnutrition develops. Prognosis is uncertain. Some cases which have come to autopsy have shown the existence of chronic Bright's disease, while some have shown cerebral tumors.

Diabetes mellitus is not common in childhood but is seen every now and then. These children eventually develop acidosis and coma, and die. The prognosis is very unfavorable and death usually occurs before the sixth year. The child is subject to respiratory, gastro-intestinal, and frequent local infections.

Cystitis is seen less frequently in children than in adults. Diagnosis and treatment vary little. Clumps of pus cells in the urine are suggestive of vulvovaginitis.

One of the common disorders met with in childhood is pyelitis. With recurrent attacks of fever, frequent urination, and thirst, pyelitis should be suspected. Examination of the urine shows a hyper-acid reaction, a varying amount of albumin, and microscopically an increased number of isolated pus cells. Pyelitis recurs and tends to become chronic. For a time the symptoms may be latent, to reappear quite suddenly. The colon bacillus is the usual cause of the affection. The prognosis is good if the condition is recognized, but should it go untreated the cortex and pyramids become involved by extension from the hylus and death may ensue. Since almost 80 per cent. of these cases occur in female children, it is necessary to obtain catheterized specimens for satisfactory examinations. At times the infection seems to be an ascending one. At others it seems to be sequent to an intestinal infection.

Pain in the loin, pain on micturition, blood in the urine, should always suggest the possibility of a tuberculous infection or a calcareous deposit in the kidney. Careful examination of the urine, tuberculosis elsewhere, tubercle bacilli in a catheterized specimen of the urine, and *x*-ray clear up the diagnosis.

Acute nephritis occurs quite commonly in children after acute infections. Especially is this true after scarlet fever and tonsillitis. In infancy congenital syphilis is the prime cause. As a rule nephritis in children is not diagnosed until the patient is dropsical and anemic, or he may show only fever and vomiting. The urine is dark, scanty, and examination discloses albumin, casts, and possibly blood. These cases as a rule are those which have been neglected during some illness by failure to watch the urine. There are, however, some cases in which no history of previous disturbances can be obtained. Consequently primary or idiopathic nephritis is diagnosed. Inflammation of the kidney, as we have seen before, is one of the disturbances so frequently associated with tonsillitis, rheumatism, chorea, and endocarditis. Chronic nephritis is occasionally seen in children, quite like the adult interstitial type.

Orthostatic albuminuria occurs not uncommonly in children and is peculiar to this age. As a rule these children are found to possess an undue or increased curvature of the lumbar spine. The child usually is brought for examination because of general ailments such as pallor, puffiness about the eyes, headaches and pains in the back, at times nausea and abdominal pain. The diagnosis is not made until after the urine examination. These cases sometimes show no albumin, especially if the child is kept in bed. But if he is allowed to stand for a period of time, the urine collected will show albumin. The condition usually passes off; the prognosis is good. Treatment is non-medicinal. The general health should be improved by exercise and out-of-door life.

Hydronephrosis is a rare condition in early life but the paranephritic abscess is not uncommon. The peculiarity of this condition is that it appears so quickly. There is a mass in the loin, a rigid abdomen, pain and tenderness and septic symptoms. Usually there is pyuria. Treatment is surgical and recovery is prompt.

Tumor growth of the kidney is sarcoma. It is not rare. It is usually slow at first, not attracting attention until the tumor has attained large size and the child has a prominent abdomen. It is not until then that the child suffers wasting and anemia, with fever and certain death. Operation only seems to hasten the inevitable.

Enuresis is one of the most disturbing conditions met with in children and requires most careful investigation and study in order that the cause may be learned and eliminated. As a rule it appears in the nervous child of bad environment and imperfect training, or in the child who has never established voluntary control. It may be due to a hyper-acid condition of the urine, to an irritation along the urinary tract, to improper diet or indiscretions in food. It may be due to bad sleeping habits such as being too warm, or it may be caused by an irritation about the perineum. In later childhood it may be due to the onset or developement of the sexual sensations. Habit and laziness seem to be quite common causes, but the possibility of other causes must be eliminated first.

Gonorrheal vulvovaginitis is a very frequent infection in females and is frightfully contagious. Some state it occurs in approximately 20 per cent. of hospital cases, and in 5 to 10 per cent. of patients in private practice. The disease may show itself by a vulvovaginal irritation or pus discharge, which may appear suddenly. In such cases it is probable that there has been former vulvar involvement which cleared up, but from which a definite cervicitis developed and persisted, although latent. Complications are rare, though ophthalmia, salpingitis, and lymph adenitis have occurred. The end-results of untreated cases are unknown though it might seem that the disease dies out before maturity is reached. The infection is gonorrheal and it may almost be said that any purulent vaginal discharge is of this infection, excepting possibly the vaginitis which sometimes accompanies measles. Treatment is by medicated irrigations, instillations, and hot douches. The vaccines have been disappointing.

It is a rare thing to find gonorrheal infection in the male infant but it does occasionally happen and seems to be mostly in institution cases, possibly carried from another child by unwashed hands or infected clothing.

The malformations of the kidney are seldom recognized unless by careful, complete examination for some obscure disturbance, just as is done in the adult.

Balanitis is a frequent condition in young male babies and is due to neglect and uncleanliness. Proper stretching, retraction and cleaning the foreskin in the newborn babies will greatly decrease the uncleanliness as well as the necessity of the far too-frequent circumcision.

Paraphimosis is the result of improper and ignorant manipulation of a phimosis. The obstetrician should instruct each mother most explicitly in the care of the genital organs. It would prevent many distressing complications.

Congenital defects of the lower genito-urinary tract are occasionally met with. These may be extrophy of the bladder, which is a most serious condition, a stricture of the urethra, or a hypospadias. Epispadias is less frequent. Hydrocele of the cord is not an uncommon condition in male babies and as a rule the condition takes care of itself. In a few cases surgical intervention is necessitated. These malformations are of course surgical.

In children meningitis usually involves the pia, and the common forms met with are tubercular meningitis, suppurative meningitis (that is, one secondary to some local infection, commonly pneumococcus), and epidemic meningitis. This probably is a diplococcus, intracellularis infection. These forms are mentioned in the order in which they are most frequently found.

Tubercular meningitis is of fairly common occurrence, and is seen in infants of six months to children of seven or eight years, most common, probably, in early life. Symptoms do not differ from those in the adult, and as in the adult it is usually a terminal involvement.

Suppurative meningitis is a secondary form following or complicating, as a rule, disease of the lungs, infection of the mastoid, and other local infections. It is more commonly seen in children than in adults, and death within a week is the invariable result.

The posterior basilar type is limited to infants, chiefly in the first year. It is sporadic in distribution and is due to a diplococcus of the meningococcic type. Possibly the infection is the same as in the epidemic type. The disease runs a chronic course with a more or less acute onset, retraction of the head, and at times convulsions and vomiting. There is opisthotonus, rigidity of the limbs, and evidently partial or complete blindness. The fontanelle is bulging and the head is increased in size. The temperature is elevated and irregular for 10 days or two weeks, then remains normal. Pulse and respiration are regular and apparently little affected. The child continues in this condition for two or three weeks, apathetic but conscious. It cries only when disturbed. Emaciation gradually takes place, food is poorly taken, and death results from exhaustion. Death, however, is not the invariable result, for some of these cases do recover showing a hydrocephalic condition and some partial paralyses. Pathological changes are noted in the spinal fluid first, and in the meninges, where adhesion takes place. Oftentimes the foramina are blocked, producing thereby an increase in the hydrocephalic condition which from pressure may eventually result in the child's death. The treatment is the same as for the epidemic form.

Epidemic cerebrospinal meningitis differs in no respect from the disease as seen in adults.

It is very common to find in infants and children a condition simulating meningitis. This is known as meningismus and may occur with any of the acute infections. It probably is due to cerebral congestion or a toxic effect upon the vasomotors.

Tetanus is rather rare in children but is occasionally seen in the newborn, where the infection evidently occurs through the umbilical stump.

Hydrocephalus is a disease peculiar to infants and is of two varieties, internal and external. The former is more common and is due to blockage of the ventricular openings. This blocking may be due to tumors, inflammations and adhesions, or congenital malformations. Where the blockage is extreme or complete, death occurs, but where only partial the child may continue to live and thrive.

The external hydrocephalus is less common and is due to a collection of fluid in the meninges. It may be caused by hemorrhage, trauma, or inflammation. Diagnosis is with difficulty but treatment is more hopeful in this form.

Intracranial tumors are infrequent but the large proportion of those found in children are tubercle formation, gumma, or glioma. These usually involve the base of the cerebrum in the pons region. Headache, vomiting, and optic neuritis are the cardinal symptoms. Other symptoms depend upon the location of the growth. The ultimate result of these conditions is death. Surgery in glioma may offer some hope, and specific treatment in gumma will probably afford a more promising result. The tubercular form is very likely to spread and involve the whole of the meninges.

Meningeal hemorrhage is not uncommonly seen in the newborn. Partial paralyses may appear and remain, but as a rule there is absorption of the clot and relief. There may be some remaining thickening and pressure, and impaired use of some one or other ofthe parts. In later life meningeal hemorrhage is seen at times with whooping-cough, with possibly a resultant paralysis such as hemiplegia. However, the most common cause of hemiplegia coming on in childhood is syphilis.

Facial paralyses are at times seen in children, the most common cause being pressure on a peripheral nerve by an enlarged node. Another form of peripheral paralysis which formerly was much more commonly seen than at present is the diphtheritic. Usually it follows an untreated infection. It is usually local, not involving more than one or two muscles.

Infantile paralysis, or acute anterior poliomyelitis, or encephalitis, is a definite general infection with selective destruction of the motor cells. Only 25 per cent., however, show apparent residual paralyses. Infantile paralysis is generally present at all times in isolated cases, especially during the summer months, but it recurs in violent epidemic outbreaks. The acute disturbance is frequently so slight and so simulating a mild gastro-intestinal disturbance that it is over-looked, and the diagnosis made only upon the sudden appearance of flaccidity in one or another group of muscles. The acute attack is accompanied with fever, vomiting, and possibly diarrhea. The child is fretful and moans or cries with pain if handled. There is tenderness along the spine and especially over the nerves and muscles of the involved part. The paralysis may select any muscle or all muscles. The most fatal involvement is that of the muscles of deglutition and respiration.

Polio-encephalitis is an involvement of the meninges and cortex with a residual thickening. Recurrent convulsions or epileptiform attacks occur, localized as a rule on one side of the face and body. The muscles do not show the wasting and atrophy seen in those cases in which the involvement is located in the cord.

Spastic diplegia and paraplegia are birth conditions, often described as birth palsies. There is usually a history of difficult birth and asphyxia, possibly convulsions. Frequently they are associated with mental defects and slow development. These cases are usually those of general body weakness, consequently intercurrent infection is the cause of death. There are, however, quite a number who live, and by the fifth or sixth year they are first able to stand and use the limbs. They may gradually develop more use, but never complete and free use. At times one finds these children very bright, especially along one or more lines. They are always more or less delicate and difficultly nourished. Treatment is extremely careful handling and feeding, with great patience in manipulation of the parts to overcome spasticity. Ingenious methods must be employed to encourage development and use.

Pseudohypertrophic paralysis is a disease, peculiar to children, which affects only the muscles. It occurs in males chiefly and appears about the sixth or seventh year. As in hemophilia, it seems to descend through the females of affected families to the male children. The first symptom noted is some awkwardness in movement, or perhaps a weakness, so that the gait is affected. The child usually is the one who has developed slowly from infancy, and walked late. He may stammer, or there may be some mental backwardness. The child is usually brought to the physician because of feebleness, awkwardness, and frequent falls. It is noticed that the muscles of the calves and buttocks are enlarged out of proportion to the other muscles, and that the patient stands with his feet far apart. If the patient is on the floor he has a peculiar manner of "climbing up on himself" as he rises to a standing posture. There may be an increased lordosis and later a talipes equinus. Ultimately the condition involves other muscles of the thigh, pelvis, etc., whereupon it may be noticed that the muscles first affected are wasting. Eventually, weakness becomes extreme. The child is unable to stand or raise himself and death occurs from general malnutrition, failure of respiratory muscles, or an intercurrent infection. This is usually by the twelfth or fifteenth year. The pathology here is replacement of the muscle cells by a deposition of fat, and later by connective tissue. There appears to be no other involvement than that of the muscle.

There are other types of muscular atrophy which are described under the name Erb and Landouzy-Déjérine. The first involves the muscles of the shoulder and arm, possibly the leg, and the second those of the face and shoulders. These are injuries to the brachial nerves, happening at birth, and recovery depends upon the degree of injury.

There is a condition of congenital muscular weakness known as myotonia congenita which dates from birth but is not recognized until the infant is a few weeks or a month old. The main feature is the general flabbiness of the musculature and the accompanying weakness. The child assumes positions impossible for a child of normal musculature. There is an appearance of general emaciation, and all tendon reflexes, also electrical reactions, are absent. Intelligence and sensation are normal. At times there seems to be some little improvement, in that the child may learn to stand or walk, but the ultimate result is death, probably from some intercurrent, possibly respiratory, disease.

There is one type of progressive muscular atrophy which is peculiar to childhood. This is known as the Werdnig-Hoffman type and begins early in life, at times seeming to be congenital. It occurs in families. The infant, usually about six months of age, gradually becomes weaker. The musculature is flabby, especially that of the upper arms and thighs, and later extending to the distal portion of the extremities, and then the trunk. There is inability to hold the head up, inability to sit or walk. Intelligence and sensation are not impaired. Death results, usually from some respiratory disorder. The lesion found is a degeneration of the anterior horn cells and their out-going nerve trunks. The clinical differences of these last few conditions are almost *nil*. In fact, one seems to run into the other. The only difference may be the pathological lesions.

The symptoms arising from a cervical rib may simulate one of these conditions but it can readily be eliminated by a skiagram.

Friedreich's ataxia is a rare condition but it always appears in childhood. There is usually a history of unsteadiness of movement, dating from early life—possibly from infancy—but well-marked symptoms do not appear until about the tenth year. The condition at times seems an hereditary fault, especially arising from ancestors who suffered from some severe nervous disorder. At other times there seems to be no such history. These children are usually backward and late in development. Usually about the sixth year or so, often after an acute infectious disease, there appears the ataxia or unsteady movement. This is slight at first, noticed probably in the upper extremities, or perhaps the head. The gait is unsteady and the legs are placed far apart. Later there is definite staggering and uncertainty about motion. There is later almost constantly the talipes equino-varus. The tendon jerks vary, probably in most cases not present. The face is dull and stolid and speech is slow, thick, and indistinct. The eyes are unaffected except that sometimes a nystagmus is present. The disease is slowly progressive and the child becomes a helpless cripple, life lasting until some intercurrent infection takes him. The lesion here is usually a sclerosis of the lateral tracts and possibly into the cerebellum.

An acute ataxic condition may appear in children of neurotic tendency, especially after acute infectious disease. This may last for a short period of time and gradually disappear after a year or two. The disturbance seems to be the result of the virus of the infection, which attacks the cortex cerebellæ.

There is a congenital ataxia which is first manifested only by a nystagmus in the first few months of life, but later all movements attempted are ataxic and the child learns to sit and stand with extreme difficulty. Gradually the child gains control and by the fourth or fifth year movements become normal. The pathology of this condition is unknown and the condition tends to recovery, although it may be necessary to educate muscular action.

Spasmus nutans is a peculiar affection of infancy which is characterized by an arrhythmic movement of the head and of the eye-balls. This motion may be nodding, lateral, or rotary. It occurs usually in infants from 6 months to 18 months of age. It has been variously attributed to rickets, to dentition, and to impairment of vision. The condition lasts rarely over three or four months and gradually disappears, as soon as the child's general health is improved.

Tetany is a condition much more commonly seen in infants and less frequently in children, and is caused by faulty feeding or gastro-intestinal disturbance. It is always seen in rachitic babies. The hands are flexed at the wrists and kept pronated, with the thumb drawn rigidly toward the little finger and the fingers held in extension at first two phalanges, while the distal is flexed. The legs are extended, feet extended, and toes held in dorsal flexion at first phalanges while the distal is in plantar flexion. The spasm may come on intermittently and the condition of the hand may be aggravated by any sensory irritation. Tapping just anterior to the ear and below the zygoma causes a contraction of the facial muscles. This condition seems to appear in those babies of a hyper-excitable nervous state and with a disturbed calcium metabolism. It disappears with the proper regulation of diet. Laryngospasm is another manifestation of tetany. At times one sees such spasmophilic conditions which are undoubtedly the result of certain protein sensitizations. Eczemas to some degree are always co-existing.

Epilepsy usually has its onset in childhood. One-eighth of the cases begin about the third year, 5 per cent. probably in the first year, and 7 per cent. during the early part of second dentition.

Epilepsy appears in families of a neurotic or insanity taint. It may be associated with malformation of the brain or it may come on after some disease or injury to the brain. It is divided into two classes called the grande and the petit mal. This, of course, is true epilepsy. There is another type known as the Jacksonian, only partial in its involvement. The chief feature is loss of consciousness. In the grande mal there is a definite aura. The child cries out preceding the attack, which is more severe and longer in duration. Afterwards there is sleep, and during the attack involuntary urination and defecation. In the petit mal the attacks are slight, without aura, and the subsequent sleep is but a drowsiness. The attacks gradually become more frequent, more severe, and eventually the brain substance suffers and insanity comes on. Medical aid by sedatives, proper dietary supervision, and careful regulation of habits may control the attacks very greatly. Syphilis probably plays a large part in a great number of epilepsies and early recognition and treatment of this condition may clear up the epilepsy and prevent the degenerative changes which would eventually end in insanity. Epileptic seizures or attacks are often diagnosed when they are only recurrent convulsions of some other disorder, usually intestinal.

Terrors, which are usually nocturnal, are sudden attacks of fright and crying with hallucinations. They usually occur in children of neurotic temperament—excitable, imaginative, and high-strung. These patients are subject to digestive disturbances and frequently are those who have suffered or are suffering from the condition known as exudative diathesis. It is frequent, however, in older children that the attack comes on after excitement and indiscretions in diet. The evening meal is as a rule too heavy.

Habit spasms are not uncommonly seen in children and usually appear with some other disturbance. For example: Snuffling and elevation of one side of the nose and lips may occur in cases of nasal obstruction.

Masturbation is much more common in childhood and infancy than is supposed. It usually begins as a result of some local irritation. It is most essential that the condition be recognized early for it is then that the habit can be best broken and the development of a sexual hypochondriac prevented.

Amaurotic family idiocy, though rather rare, is a disease peculiar to infancy. It affects only those of the Jewish race and seems to appear in certain families. There are no other known etiological factors. The infant seems normal until about the sixth month when it is noticed that the child is not developing normally. He is unable to hold the head erect and does not sit up. Also there seems to be impairment of vision and the child is little interested in its surroundings. These children are often fat and seemingly well-nourished up until the onset of the symptoms. The muscles are flabby and there is a slight increase in the reflexes. Gradually the child begins to emaciate and lose weight. A characteristic feature is the obliteration of the macula lutea by a milky or white area with a bright cherry-red spot in the center. There is atrophy of the optic discs and the condition is found alike in both eyes. The wasting and general weakness proceed and usually before the second year of age some intercurrent infection causes death.

Mental deficiency may be, as we have seen, secondary to some other disorder. It may also be an apparent primary condition. Naturally enough both types are brought frequently before the pediatrician.

These conditions are peculiar to early life. The etiology is imperfectly understood. As a rule, however, the mentally deficient child is a descendant of a mental deficient. Other causes frequently mentioned, and with good reason, are parental syphilis, chronic alcoholism, impaired health, especially of the mother during pregnancy, and unfavorable surroundings. The pathological changes may be atrophy of some portion of the brain or under-developed convolutions and sulci. Microscopically there may be degeneration or an absence of ganglion cells. Mental deficients are classed as a rule as idiots, imbeciles, or morons. Any division, however, is only arbitrary and but expresses the degree to which the child is affected. The abnormality or deficiency can only be determined by comparison with the normal infant and child of the same age. It is also necessary that the mental deficiency be classed definitely so that an accurate prognosis be made for the placement and care of this unfortunate cripple.

Mongolian idiocy is a form of mental deficiency recognized abruptly by the physical characteristics. The etiology of the condition is obscure but includes etiology of general mental deficients, with the important addition of the age of the mother, since these unfortunates are usually born to a mother at about the time of the menopause, or is the last of a number of child-bearing pregnancies. The brain is small and under-developed throughout. The child has the peculiar Mongolian facies, eyes are set widely apart, the palpebral fissure is narrow and slants downward. The head is small, the child is underheight, the hands are short, especially the fingers, with the little finger markedly dwarfed. Musculature is poorly developed and joints are extremely freely movable, so that all sorts of contortions may be easily performed. The child is a mouth breather, tongue is large and protruding, and the lips are excoriated. There are frequently other malformations and defects. These mongols are very backward and they have little resistance to disease and infection. As a rule they die before the age of 10 years.

Chorea is a functional nervous disorder which appears in children, usually after the seventh year, and is probably twice as frequent in the female. It is characterized by irregular movements of any part of the body. These are aimless and involuntary but involve only the voluntary muscles. The disease is closely related to rheumatism and associated with tonsillitis, arthritis, endocarditis, poor nutrition, and anemia. There are, however, quite a few cases without this relationship except for the poor nutrition and anemia. They usually occur in the highly strung neurotic female with a high degree of mental alertness and an over-abundance of energy for study. The attack may come on after any acute infection, or as is frequently the case, after the winter school season. Fright has been known to cause the onset, as well as an imitation of others suffering from the same disorder. It is a precursor of a neurotic, hysterical individual and frequently attacks children about the age of puberty. Its prevention can quite readily be accomplished by proper care of the "nervous child" by avoiding too close application to study and guarding against poor nutrition and anemia.

The skin at birth is a tender red covering which about the second or third day displays a yellow cast. This disappears usually about the fourth or fifth day and is physiological. Its persistence, with deepened color, is the icterus neonatorum rather frequently observed. By the end of the second week of life the infant has desquamated its original epidermis unnoticeably, unless carefully sought for. The sweat glands are not active until the third or fourth month, and perspiration is not abundant during the first year. Should it be present it suggests rickets, or general weakness in acute disease.

Ichthyosis is a fetal disease of the skin. It is rare and is characterized by a thick, scale-like epidermis not unlike parchment. Where the involvement is severe the life of the child is greatly endangered. The severe degree involves the body orifices, with contractions of the skin. The epidermis is thrown off in scales, which soon re-form. Milder forms do not impair the child's life. The "alligator boy" is an example of ichthyosis. The condition may be improved by persistent use of oils and baths.

Miliaria, or obstruction of the sweat glands, is a frequent condition in infancy. It is non-inflammatory and usually disappears shortly.

Seborrhea is a functional disease of the sebaceous glands and peculiar to infants. It is commonly seen on the scalp. Untreated it may cause inflammatory or eczematous disturbances. Local treatment is all that is required for its removal and prevention.

Eczema is not only the most frequent but the most important disease of the skin in early life. In older children eczema is the same as that in adults. In infancy several conditions predispose to it. They are the tender skin, and the abundant gland secretion; the prevalence of digestive disturbances; and in exudative diathesis it is one of the chief manifestations. Eczema appears in certain families more than others, and it usually appears in the fat, apparently healthy child rather than in the poorly nourished. These children usually show some sensitization to vegetable or animal protein. A diet rich in fat or a diet of high carbohydrates may oftentimes be a cause. Infection is secondary in involvement and importance. As a rule the disease begins upon the cheeks as small red papules which spread, coalesce, and ooze serum. Crusts form and are removed by scratching, or rubbing of clothes, to leave a raw, bleeding surface which becomes infected. The child's health may be maintained, but frequently the constant irritation disturbs the rest and eventually undermines the nutrition. Local measures are indeed of advantage, but only when accompanied with general treatment, especially of the gastrointestinal tract. It is frequent, however, that it persists in spite of treatment, until the child attains the age of solid diet.

Other forms of eczema frequently seen are those from exposure to cold and other irritants, chemical or mechanical.

Intertrigo is probably a form of eczema, seen on the moist surfaces, but may be a mere erythema in the skin folds. It is oftentimes seen as an erythema, later red papules, vesicles, and pustules, as in cases of ammoniacal urine. The skin under the diaper is at times frightfully burned and is a raw bleeding surface. The only necessary treatment in this instance is a change of diet to prevent the strong alkalinity of the urine.

Furunculosis is commonly seen among infants of poor nutrition. The skin and subcutaneous tissue is poorly resistant to infection and the local abscesses spread from place to place. Frequently a cellulitis develops, especially if the scalp is involved. Both local and general measures are essential.

Impetigo contagiosa is a disease appearing chiefly in children, readily communicated from one to the other. The disease appears first as a discrete vesicle, which becomes purulent and breaks, leaving an irregular ulcer. New vesicles form and the condition spreads. It is often seen in institutions, in children of a school, or among those frequenting public swimming pools. It is very responsive to treatment.

Urticaria is frequently seen in early life. The wheals are usually associated with papules and vesicles, and scratching infects and causes pustules. The child's skin is extremely susceptible, which accounts for the variety of lesions and the increased intensity of the attack. The onset is abrupt and the most frequent cause is an intestinal upset. Local treatment to relieve the symptoms, and general treatment to remove the exciting cause, are to be employed. Urticaria is often seen in those children who are especially sensitized to certain proteins, Violent vomiting with an asthmatic or anaphylactic-like attack is oftentimes associated.

There is also a petechial scarlatiniform eruption frequently observed in some acute intestinal disturbances. There may be fever, vomiting, and diarrhea, but no respiratory symptoms. The rash quickly disappears upon the thorough evacuation of the bowel and the child returns to normal. It is quite a cause of alarm because of its resemblance to scarlet fever. Frequently, too, this rash may simulate that of measles and be difficult to differentiate.

Scabies is frequently seen in children and less frequently in infancy. It comes from association with others so affected, or through infected towels or clothing. Its diagnosis and treatment are the same as in adults. In children, scabies may cause a variety of rashes.

Ringworm of the scalp is seen frequently in children in institutions. It is often epidemic there. It may appear in other locations than the scalp where it has the appearance of tinea circinata. The diagnosis and treatment is common to that of tinea tonsurans.

Erythematous rashes very often appear after certain drugs.

The most common peculiarities met with in diseases of the osseous system of the infant are those of deformities, malformations, or developmental defects. Disease in the infant is oftentimes accompanied with some manifestation of skeletal involvement. In rickets, for instance, the embossing of the skull, enlargement of the epiphyses, and the bowing of the long bones. The normal growth areas persist in their cartilaginous or membranous character. Very early changes in these areas are suggestive of syphilis. Other reasons for their early conversion are poorly understood.

At times the infant is born with very fragile or brittle bones. Fractures occur even in intra-uterinal life. This seems to be the result of some defect in the cartilage or bony covering and the prognosis for recovery is much better if the fracture occur some little time after birth rather than multiple fracture about the time of birth.

Osteomalacia of infancy appears about the time the child begins to walk or a little later. The cause is absolutely unknown. At times a similar condition has seemed to follow infectious disease. The bones are soft and rarified although the periosteum is thick. Any weight or pressure, such as one leg over the other, will cause a deformity of the bone. The trunk may distort the spine and the pelvis be twisted out of shape. The bones of the shoulder girdle may suffer. The general musculature of the body suffers and treatment is essentially the same as that of rickets. This disease has been classed by some writers as tardy rickets.

Achondroplasia is a rare condition found in certain children with deformities and is most evident in the femur, humerus, and bones of the skull. The long bones seem to suffer from lack of growth in length, so that the shortened limbs appear greatly out of proportion to the length of the body. These patients are especially fat and the fat lies in folds. As a rule there is a marked lordosis and a prominence of the belly. The hands are short, broad, and thick and the fingers separate producing the so-called trident hand. The upper extremities are not always involved. The head is large and broad with a full forehead, but the face is small, nose flat and depressed, lips and tongue thick. The mental condition seems unimpaired, as does the general health. These patients are at times confused with cretins and mongols. The condition is sometimes spoken of as fetal rickets. It is one of the chief causes of dwarfism. Its etiology is unknown, although there seems at times some hereditary association. Treatment is to maintain general nutrition. Glandular therapy has been advocated but there seems to be little chance of anything efficacious unless it be directed to fetal life.

In infancy, tuberculosis of the bone is rare, unless it be a dactylitis. Syphilis of the bone is seen not at all infrequently. Very early it is seen in its destructive stage, while later, at the usual time these cases come under observation, it is in the productive stage.

Scurvy causes subperiosteal hemorrhage and bone tenderness. At times there is epiphysitis, which may become suppurative.

Rheumatoid arthritis, or Still's disease, is sometimes seen toward the end of the second year. It is a rare condition with symmetrical swelling of the joints, especially the fingers, little fever, and no pain. Spleen and liver are both enlarged and general nutrition suffers.

Rheumatic nodules are quite common in older children who suffer rheumatic attacks. These nodules appear often about the bony prominences of the joints and frequently over the cranium. They probably are isolated streptococcic infections accompanying acute conditions.

Congenital dislocation of the hip is probably the most frequent of all congenital dislocations. One or both hips may be involved and the condition may be of a hereditary nature. Girls seem much more often affected than boys. The real cause is uncertain.

Congenital syphilis is an intra-uterine infection of the fetus from the mother. At times the disease manifests itself at birth or shortly thereafter. It may, however, not be manifest until childhood. At birth the syphilitic child often shows snuffles, malnutrition, erythema of the whole body or excoriations about the nose, the corners of the mouth, and the anus. There may be marked peeling of the skin of the palms of the hands and the soles of the feet. The infant, as a rule, has somewhat the appearance of "a little old man." These skin lesions may not appear until sometime during the second month of life. Again, it may show itself as an acute multiple epiphysitis with bony destruction. It may evidence itself by hemorrhages. Superficial ulcers of the skin, of the lymph-nodes, and of the mucous membranes of the mouth, which tend to become subacute or chronic, are manifestations of syphilis. The spleen, as a rule, is quite noticeably enlarged and easily palpable. Later in childhood there may be only chronic snuffles, possibly bloody; there may be only persistent malnutrition; there may be lymph-node destruction and "punched-out" ulcers of subcutaneous tissues; there may be bony nodular enlargement or the so-called "saber shin;" there may be the characteristic Hutchinson's teeth; there may be convulsions with central blindness, deafness, or in fact, any muscular or sensory paralyses. Wassermann reaction shows positive results although at times persistent negative Wassermanns may be obtained, becoming positive only after the institution of treatment. A detailed family history, a detailed history of the birth conditions and of the first few months of life is most essential lest the diagnosis of syphilis be overlooked.

Tuberculosis in infancy is probably much more frequent than is generally supposed, and death due to it is oftentimes attributed to some other cause. The infection is mostly respiratory and the disease manifests itself either in bronchitis, in broncho-pneumonia, in adenitis, or in meningitis. Persistent loss of weight, malnutrition, and death form the usual end-results of undiagnosed tuberculosis. Unlike the pulmonary involvement found in adults, the infant lung shows isolated foci of infection, at times grouped, the younger near the older. It is not infrequent that the child suffering with malnutrition or an apparent gastro-intestinal disturbance develops a bronchitis or a broncho-pneumonia and death ensues. The tuberculous infection was probably present prior to the other disturbances but was "lighted up." Again, at times one finds a meningeal involvement which was probably secondary to tuberculosis elsewhere. But it is frequently noticed that the large, well-developed breast fed baby becomes ill, possibly with only a little gastro-intestinal disturbance and slight fever. The child becomes drowsy, turns his face into the pillow in order to protect his eyes from the light, and his fretting is replaced by a cry when he is handled or disturbed. Gradually the neck becomes rigid and the spine seems stiff, pupils dilate and fontanelle bulges, the head is retracted, the cry becomes a moan, the temperature rises higher and higher and death comes within three weeks from the onset. Whether or not this is a primary or a secondary infection is disputed. It appears that in such a typical case the infection has been direct and primary.

Tuberculosis of the lung as a rule in infancy is but a bronchitis or a broncho-pneumonia and is very infrequently diagnosed. Miliary tuberculosis is seen at times and more frequntly in infancy than in later life. Diagnosis frequently is not made until autopsy, although *x*-ray or stereoscopic examination would offer an undisputed diagnosis.

The tracheobronchial lymph-nodes are frequently involved in infancy. This is secondary to involvement in the lung and diagnosis is oftentimes not made unless there be some symptoms of pressure or suffocation. *X*-ray examination discloses masses in the lung hiluses. Tuberculous adenitis is not so frequent at this early age and it is very likely that a great many of the so-diagnosed tuberculous glands have been chronic infections of some other nature. In infancy the Von Pirquet tuberculosis test is of vastly more importance than in childhood or later life.

Tuberculosis in older children is more frequently of the bones or lymph-nodes, although tuberculosis of the lung is not at all an infrequent finding. As in later infancy, one finds also peritoneal infections every now and then. The pulmonary involvement differs from that in adults in that it seems to be basal instead of apical.

Tuberculosis of the bones involves most frequently the spine or the upper end of the femur. It is a destructive process and the onset as a rule is referred to some definite injury. In the infant the bones most usually involved are those of the fingers, with the typical dactylitis or spina deventosa.

Chronic ear disturbances are oftentimes due to tuberculosis. In all probability by far the greater part of tuberculosis in early life is due to the human type, and the bovine type is not so frequent as has been supposed.

Latent tuberculosis is oftentimes brought out by the acute infectious diseases, especially measles and pertussis.

Erysipelas is frequently met with in infancy. Early life seems much more susceptible to it. Character, course, and treatment are identical with that in adults. Prognosis is favorable unless the infection be in the infant of subnormal health.

Typhoid fever and malaria are found in infancy as in adult life. Acute rheumatic fever is seen frequently in children but very infrequently under four years of age. The only marked difference that might be mentioned is that pain may be complained of over the shafts of the bones rather than at the joints. However, articular involvement is seen and presents all the cardinal symptoms.

Influenza, contrary to much common opinion, is frequently found in infancy and childhood. The nature of the disease is essentially the same as in adults. There is, however, often observed the meningeal type, in some cases being only toxic, while in others true influenzal meningitis. Probably the intestinal form of the disease is more frequently met with in early life.

The contagions naturally are more frequently seen in childhood and in every sick child it is the duty of the physician to look for and eliminate contagious diseases. Other than that the child is more susceptible—in other words, has not established an immunity to these contagions—and that the disease is more severe at this time, there is no difference from that seen in adult life. The younger the child, naturally the more dangerous the disease.

CHAPTER XVII

PROPHYLAXIS AND TREATMENT

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GENERAL PROPHYLAXIS

The branch of medicine which offers the greatest possibilities is, probably, that of prophylaxis. All over the world experiences of the recent past have clearly demonstrated the dire results of conditions that were in great measure preventable. This statement refers more particularly to the physical condition of the adult male throughout the world as revealed in examinations for the selection of the fighting forces. While there were many rejections for defects that were detected at the time of physical examination, many young men after acceptance were found to have defects which rendered them incapable of withstanding the ordinary routine of the soldier's life.

The reason for this was primarily the absence of organized effort, fostered by wise legislation, directed to the proper physical development of the young. Education of the public and efficient legislation are the prime factors in a purposeful compaign seeking the development of a vigorous race and the prevention of disease. The physician is in a position to be of great service in directing the enforcement of health laws and to serve in an advisory capacity in the framing of legislation. In this relation, the physician who devotes himself to the problems of childhood may render the greatest service, since it is in the early years of life that the greatest benefit can be attained through the prevention of diseases and unhygienic practices which may result in organic defects.

Prophylaxis should aim at (1) the preservation of the normal, and (2) the prevention of the abnormal. In order that a healthy race may be developed it is essential that the parents be healthy. In the prenatal period it is possible to remove many contributory causes which have direct bearing not only upon inherited disease but upon hereditary tendencies and constitutional disturbances. Much can be accomplished by diminishing the number of injuries incident to parturition. This period naturally belongs more to the domain of obstetrics, but the legitimate pediatric interest in the newly born extends in a measure to intra-uterine life.

The diseases of the newly born, it should be emphatically stated, belong to pediatrics. Immediately after birth the infant often continues under the ostensible care of the obstetrician, with the natural consequence that the obstetric interest in the mother deprives the child of the special consideration which it would receive were it under the care of a trained pediatrist. Only when obstetricians themselves recognize the wisdom of having the pediatrist assume charge of the newly born at birth, will adequate benefit accrue to the infant from the point of view of prophylaxis. Infancy—the time from birth to two years—offers a wonderful opportunity for the practice of prophylaxis, in view of the fact that then the foundation of the individual health is laid. Many nutritional disturbances have their inception at this period, and these have not only immediate but also remote effects. At this developmental period all the systems of the body are evolving and careful supervision is necessary for their proper development. Although dentition occurs during this period, it should not, as is often the case, be held responsible for every ailment, since by this flagrant error, prophylactic measures are frequently defeated.

Close observation and a thorough knowledge of the normal disclose many evidences of disease. Not only may already existing conditions be recognized and promptly remedied, but through proper prophylactic measures many deformities and diseases may be averted. The "neglected period," or period of independence, is that from the second year of life to school age. At this stage it is difficult to supervise the child closely owing to the comparatively greater freedom that it is given. The molding of the character, inculcation of respect for authority, and prevention of bad habits should receive strict attention during this period, when hygiene and environmental influences also play an important role.

During the "age of exposure," or school age, prophylaxis must be exercised to even a greater degree, continuing up to puberty. The regulation of habits, rest, periods of study, recreation, diet and exercise, should all be given a fair measure of consideration. No less important is the correction of postural defects, not only in respect to the child's posture out of school, in exercise and athletics, but also in the classroom. Infections may often be averted by due consideration of sanitation even though the dangers of exposure are greater in this age-group than at any other time of life. Toward the end of this period, namely, at the time approaching puberty, a proper education in matters of health and sex will greatly benefit the prospective parents of the coming generation.

GENERAL TREATMENT

The tendency of our age is toward the prevention rather than the cure of disease. The necessity for cure arises however, and a thorough knowledge and application of treatment are essential.

When normal physiologic limits are transgressed through the influence of extraneous chemical or physical stimuli, disease results. Recognition of such influences is a necessary preliminary to the application of proper therapeutic measures for the restoration of normal conditions. Stimuli may act by increasing or diminishing vital energy, the ease of accomplishment being modified by many factors, including environment, age, predisposition, familial tendency, habit, and the effects of disease, and these may to a greater or lesser degree also influence the strength of the reaction to the therapeutic stimuli.

In the treatment of disease in the young the coöperation of the parent or guardian is essential to success. Through the education of the public cases of illness are now brought to the attention of the physician earlier in their course, which helps to avert the development of many permanent injurious sequels. It should be remembered from a therapeutic point of view that there is a wider fluctuation of function in the infant than in the adult, and that its regenerative power is also greater.

The aim of all treatment is to restore normal conditions, which may be accomplished either by drugs or other remedial measures. In the administration of drugs not only the dosage and therapeutic qualities are of importance, but the proper application of a given drug to a particular condition must be considered, as well as the reaction of the infant toward the drug, with reference to predisposition and possible idiosyncrasy. Any familial tendencies and predispositions should be learned by securing a careful family history before prescribing, in which connection it is wise not to pass over lightly any suggestion or hint given by parent or guardian. The combined constitutions of the two parents may result in some peculiarity in their offspring, hence, each patient should be considered as an individual.

During the various periods of development, there are stages in which certain systems are especially sensitive to therapeutic agents, hence, these should be prescribed with caution as even transitory effects may cause untoward sequels. Especially is this true of functional conditions of the nervous and digestive systems.

Environment and heredity play an important rôle in the predisposition of a patient in regard to both functional and organic disturbances. Environmental factors particularly predispose to the functional disturbances which if prolonged may effect irritability of a system and result in chronic disease. Heredity may predispose not only to functional disturbances through abnormal influences upon certain organs and systems, but also to organic disease. That the ill effects caused by continued abnormal conditions may be lessened, great stress must be laid upon the necessity of a careful history and a thorough primary examination of each patient.

In this way only is it possible to determine the therapeutic measures necessary to produce most promptly the desired results. Beside these, many measures may be employed which in themselves do not cure disease but aid the return to normal. While such measures may be of invaluable assistance their over dosage or indiscriminate use may result in untoward effects, and possibly the substitution of another condition in the place of the one it is sought to overcome.

CARE AND NURSING

Proper care and nursing during illness are as essential as and often more vital to the outcome of disease than the use of drugs. In the care of the sick child, its comfort must be first considered. The child should receive a daily sponge bath unless this is specially contraindicated. This should be given in bed. The bed should be kept fresh and clean and free from any irritating substances, including crumbs and food particles, and should be warmed when necessary.

The mattress should preferably be of hair; it should be covered with a rubber draw sheet over which should be a bed pad and sheet. The pillow should be of hair and should support the head and shoulders. It is superfluous to add that the sick child should be in a bed by himself.

The underclothing and diaper should be of some absorbable material to prevent the chilling of the surface of the child, and should be changed whenever wet or soiled. The feet should be kept warm, which implies that in the case of an infant, socks or stockings are necessary. The overclothing should be loose and should allow free movement of the patient, especially in those diseases in which any restriction impairs the child's comfort. This is particularly true in pneumonias, in which there is usually a tendency to overburden the child with coverings so that the difficult breathing is further hampered.

It is important to change the position of the patient at intervals so as to forestall the development of bed-sores. The skin should be kept clean and dry, especially about the buttocks and groins and other folds where intertriginous ulceration or other skin lesions may develop. In washing the skin, bland soaps and clean water should be used, and soft towels should be employed with care not to injure the skin in drying. Dusting powders and mild ointments and astringent lotions are at times indicated.

During illness the condition of the mouth and teeth are not normal so that their hygiene is necessary for the comfort of the patient.

Particularly there should be no excitement of the patient, and the desire of the anxious parents to have these small individuals go through their little tricks is at times detrimental to their welfare during illness.

Rest and sleep are essential, and except for taking of nourishment, and in some cases of medication, the patient should be allowed to sleep undisturbed. A child should be kept in bed during a febrile period, and afterwards for a length of time sufficient to assure progress in convalescence and to so restore strength that the body waste incident to exercise will not encroach upon the stored energy and thereby retard return to full health. The return to the routine of exercise should be gradual, the patient being permitted at first to sit up in bed, then out of bed in a chair for a limited period, and to walk only when the strength has fully returned.

Attention should be given to the bowels and kidneys and careful records made of their functional activities. The stools should be kept for the inspection of the physician and the urine should be examined from time to time when indicated. There should be at least one evacuation of the bowels daily unless especially contraindicated as, for instance, in appendicitis.

The rate of respiration, pulse and temperature should be recorded at stated intervals, remembering that the only reliable reading in the infant and small child is the rectal temperature. The most dependable information about the respiration and pulse is obtained during sleep, especially of the very young child.

At the onset of illness, especially where there are other children in the household, a child should be isolated in a room which contains no hangings or other material. In this way cleansing and disinfecting of fabrics and hangings is rendered unnecessary. The patient should be isolated until found to be free from communicable disease.

AËROTHERAPY

Airing or aërotherapy is a phase of therapeutics regarding which greater appreciation is developing among the laity and the medical profession. The course of many diseases may be favorably influenced by judicious airing, while frequently disastrous terminations result from its improper employment. Not only is it essential in disease but in health as well, because certain diseases are easily acquired when other predisposing influences exist in addition to bad airing. With increasing knowledge of the advantages of fresh air many advances have been made in its application to therapeutics. In relation to the heat regulating apparatus of the small infant, the increased task of maintaining body temperature, owing to the relatively large body surface, must be borne in mind.

The requirements for ventilation of the nursery and sick room will be influenced in a measure by the age of the patient, his state of nutrition, power of resistance, and the character of his ailment. Under no circumstances should the room be allowed to be filled with relatives, friends, or inquisitive individuals, as this only deprives the patient of that to which he is justly entitled from the air, further pollutes the air by exhalation products; and lessens the chances of recovery, or at least retards convalescence. For obvious reasons, no other child should be allowed in the sick room.

The sick room should be one easily ventilated and preferably above the ground floor; it should be selected with a view to the greatest possible amount of sunlight.

The windows should be so situated as not to cause draughts, especially over the patient's bed. The amount of ventilation will naturally depend partly on the atmospheric temperature, it being desirable to maintain a uniform temperature in the sick room of from 65 to 70°F., unless otherwise especially indicated. The season of the year is therefore an important factor. The amount and method of ventilation will be frequently influenced by the character of the ailment, especially if the child is running about. If the ailment require-rest in bed, protection from draughts is more easily assured, but if the child is walking, standing or playing about, it may be necessary to keep the windows closed.

The furniture should be simple, so that it may be easily cleaned and disinfected. Linoleum offers a good floor covering because of the ease with which it may be cleansed. The room should not be swept while occupied by the child but the dust should be removed with a damp cloth so that particles of dirt are not inhaled by the patient. When necessary screens should be used about the bed. Gas stoves and heaters in the room itself are objectionable. The nursery should not be utilized for drying clothes.

The newer systems of heating cannot compare with the open wood fire or coal-grate fire. In planning a house provision for fireplaces should always be made. The fire may serve the double purpose of providing heat to regulate the temperature of the room, and, if indicated, as a means of drying the atmosphere when too moist.

Whenever possible, two rooms, one for the day and another for the night, should serve as sick rooms. This allows airing of one while the other is in use. In changing the patient from one room to the other, the latter should be heated to approximately the temperature of the room that is being vacated. A child needs about 1000 cu. ft. of air, and an extra allowance should be made for the attendant.

During convalescence the transition from the sick room to out-of-doors should be accomplished gradually. Naturally the patient should be adequately protected by clothing to meet the change in temperature. The duration of the outing should be lengthened from day to day and the outing should be preferably between 10 a.m. and 3 p.m., in other words, after the sun has dried the atmosphere, and before the chilliness incident after sun set has begun. In windy, dusty or damp weather or when there is a snow storm, or when the temperature is low, indoor airing is preferable. For this, the child is prepared as for going out of doors, the windows being opened more and more every day; an infant or small child may be put in the carriage or go-cart and rolled about. Indoor airing is advantageous as a stage in the gradual return to outdoor airing, especially in very delicate children.

Hot air is rarely employed in the treatment of children's diseases and never in infants. The best method of applying hot air is in the form of a bath (Turkish bath). Hot air from the heating system is injurious to sick children, particularly as the atmosphere is rendered too dry.

Cold air has within recent years come to be recognized as a beneficial therapeutic agent in the treatment of pneumonias, more particularly of the lobar type. Many clinicians, however, consider it disadvantageous in the secondary type or bronchopneumonias. Cold air, not too extreme, is also a valuable adjunct in convalescence from many respiratory diseases. In cases in which there is a possibility of reinfection, as in grippe and other respiratory diseases, the employment of the two room plan is preferable to the cold air treatment. The atmosphere of the rooms must be kept of uniform temperature and not too dry.

Moist air is indicated in diseases of the respiratory tract, when the catarrhal inflammation is in its dry stage. It is more particularly indicated, in combination with drugs, in catarrhal laryngitis, measles, etc.

Dry air is of advantage when prescribed as "a change of climate;" that is, when the dry air is to be found in a particular climate, and not when artificially produced.

Certain conditions improve remarkably under the influence of fresh air. The effect of fresh air on the delicate child has already been alluded to. In cases of habitual loss of appetite, the proper regulation of airing is often sufficient to start improvement.

Last, but by no means least, whooping-cough requires fresh air. Children affected with this disease are really more entitled to the use of parks and airing places than the healthy children. In whooping-cough the patient coughs more frequently when indoors, and at night. It must be recognized that this disease is very contagious, and because of its complications and predispositions, the healthy children should be safeguarded from the dangers of infection. Yet, because of the invaluable therapeutic advantage of fresh air in this disease, the health-board regulations should be so adjusted as to keep the healthy children away from those infected, rather than the reverse. An easy way would be to require whooping-cough patients to wear some sign indicative of infection.

HARDENING

In spite of the advance in knowledge regarding the care of the child and its treatment in disease, parents, guardians, relatives and friends often pride themselves on their success with practices contrary to the recognized principles based on knowledge acquired through extensive study and clinical experience. While the death rate of almost every community is much lower today than 20 or 30 years ago, this is due to the application of newly acquired knowledge, and not to the present mode of life, which would really tend to have the opposite influence. These statistics, though constantly improving, would be further augmented if the vicious habit of hardening were eliminated.

By hardening is meant the attempt to increase the child's resistance by exposing it to changes in temperature and atmospheric conditions in insufficient clothing. This applies particularly to the lower extremities, as exemplified in the use of socks and low shoes instead of stockings and high shoes, thus allowing the legs to be uncovered. In infancy one of the most frequent causes of colic is cold feet. Adolescents wear stockings, and when the weather is inclement gaiters or high shoes. Why, then, during the period of growth and development, should progress be impaired or handicapped by diverting the energy required for growth and development vicariously to the purpose of maintaining body temperature?

Prophylactic measures are applied in hundreds of cases merely to conserve the possible one, as it cannot always be known which one requires the special attention. These measures should also be applied in connection with so-called "hardening." While it is true that many survive this process, not because of the hardening but by virtue of the law of the "survival of the fittest," the constitutions of many more are thereby weakened and undermined, even if health is not permanently impaired or life lost.

CLIMATOTHERAPY

In prescribing a climatic change, a general knowledge must first be obtained of the pressure, temperature, humidity, circulation of air, amount of sunlight, and cleanliness of the atmosphere of a particular climate. Atmospheric pressure may be normal, as at sea level where it is 15 lb. to the square inch, or it may be diminished, as when the air is rarefied or diluted, for instance at elevations above sea level.

When the air is diluted the diminution of oxygen results in the quickening of the pulse rate and deepening of respiration. Experiments have shown that at high altitudes the blood contains an increased number of red blood-cells. Sudden changes from a climate of normal to one of markedly diminished atmospheric pressure should be avoided because they may produce alarming symptoms, as dyspnea, dizziness, nausea, weakness and syncope. It must be remembered that high altitudes are always dry and consequently there is more rapid evaporation and a greater water loss than at sea level.

The effects of temperature, circulation of air, and humidity are more or less interdependent. The circulation of air in nature is dependent upon atmospheric temperature. Circulation of air is essential to health as it favors evaporation and heat regulation of the body. This serves to explain the necessity for good ventilation. Climates with breezes such as are found at the seaside and in the mountains owe their therapeutic value in great part to the circulation of air. Relief from the oppression experienced on still days may be had by driving and the use of fans.

Extremely hot or cold climates, particularly when humid, have a depressing effect, and extreme heat alone undoubtedly has its influence upon decrease of resistance, particularly in infancy. Cold, when dry, has a stimulating effect as it promotes greater activity of the metabolic processes which results in the improved nutrition. Moist atmospheres are enervating, especially when warm. Warm air in itself is depressing, while cold air is tonic. Dry air is also tonic and stimulating.

Cold damp air is injurious, especially in infancy and in kidney disease in which the kidneys are overworked because of impeded evaporation. Warm dry air, as in the southwestern part of the United States, is stimulating and beneficial. Abnormally dry and artificially produced warm air favors irritation and infection of the respiratory mucous membrane owing to excessive evaporation of moisture. It also produces a concentration of fluids in the tissues and organs of the body.

Frequent atmospheric changes in temperature, humidity, and circulation cause beneficial effects by stimulating the human heat-regulating mechanism. The combinations of heat and humidity which are most likely to be harmful are cold damp air, warm moist air, and very dry air particularly if produced artificially. By the wearing of proper clothing greater variations in the combinations of temperature and humidity may be better tolerated and the possibility of injurious effects minimized.

Sunlight plays an important part in the treatment of disease and should be considered in selecting a climate for therapeutic purposes. It has chemical, bactericidal, and many other effects, not to mention a psychic influence. There are conditions, only a few, however, in which sunlight is contraindicated, as for example: Pellagra. Excessive sunlight sufficient to produce burns on the skin may be injurious, especially in conditions in which the function of the skin is essential.

Cleanliness of the atmosphere should always be advocated. The ill effects of air polluted with gases, or air contaminated with dirt, dust or bacteria, need no comment.

With the foregoing fundamental knowledge regarding the climatic essentials, their application in health for the prevention of disease, and in disease to promote resistance and hasten and stimulate convalescence, may be influenced by certain factors which may or may not be overcome. Naturally, social conditions are of prime importance. The well-to-do may have the advantages of climatic therapy in its widest range, while the poor, those who are more frequently in need of the benefits to be derived by climatic therapeutics, are unable to receive them. The surroundings, hygiene and nourishment, and the exposure to disease incident at crowding and bad ventilation render the poor fit subjects for this therapeutic measure. Provisions are made in many of the larger cities for outings and camps for the needy poor, but many not in extreme circumstances do not receive these advantages. At best vacations in camps are of short duration and though they have their beneficial influences in many the improvement is only transient owing to a return to the previous conditions.

The appreciation of the value of open air and the use of parks and dust-free playgrounds in cities bring within reach of the vast majority favorable climatic conditions at home. It is a poor community indeed which does not provide recreational places for its inhabitants.

In recommending a climatic change consideration must be given to the age of the patient, the present condition, the effect of removal, the means of transportation, the inconvenience of travel, the choice of climate and existing conditions at the destination.

It must be remembered that changes to extremes in climates, particularly in regard to altitudes, should be avoided. In older children the factors to be considered are the condition of the patient and the effect of travelling, including the inconveniences, dust, defective nourishment, discomforts, jarring and excitement, and the means of transportation.

The existing conditions at the destination selected must be given serious thought, more particularly with regard to the possible effect of the climate upon the individual case. Weight should also be given in the consideration of excitement, quiet and health in the community, the water supply, the quality of food, housing, baths, and medical attention.

Changes of climate may be advocated during health as a preventive measure, during acute or chronic illness or during convalescence as a therapeutic factor. Certain familial predispositions may be lessened, certain diseases rendered less severe and their course shortened, convalescence hastened, and cure more thoroughly established by the proper climatic influences.

The seashore has its advantages as have also the mountains and hills. As to the former advice should be given with reference to the proper regulation of baths, which should not be too frequent nor too long, nor should they be allowed until the individual is acclimated. Contraindications to their employment are shivering, cyanosis, discomfort, nervous excitement, sleeplessness, headache, etc.

In the mountains and hills exercise should not be taken until the individual is acclimated, when walking trips or drives through the woods may be allowed. These will have a quieting and restful influence if properly regulated.

The beneficial effects of a change in climate are noticeable in healthy school children who spend their vacation either at the sea coast or in the mountains. It is probably a good plan to alternate the vacations at the seashore and in the mountains, in this way subjecting the body to different climatic conditions and allowing the children the recreational advantages and the cultivation of independence of both seaside and mountains. Seabaths with exposure of the skin to the air are of decided benefit if not overdone. After such vacations children seem to return to their studies with renewed vigor, their physical improvement is marked, and their mental resourcefulness more developed.

DIETOTHERAPY

The dietetic treatment of the sick child is one of the most important measures at the command of the pediatrician. Many diseases are rendered obscure by the improper application of dietetic measures; errors frequently lead to the absorption of toxic material from the gastro-intestinal tract, and at times to actual production of gastro-intestinal complications. In disease there may be defective and there always is diminished digestive power.

Nature apparently serves to protect the sick against dietetic faults, which is manifested by disinclination for food or loss of appetite. Exceptions occur in indiscreet children and these pay the penalty for their indiscretion. A good example may be cited of one of the most commonly acknowledged truths as to nature's watchfulness. It is conceded that breast-fed babies pass through the period of dentition with less disturbances than bottle-fed babies. This statement is subject to attack as most careful pediatricians experience no more inconvenience with bottle-fed infants than with breast-fed babies. But to continue the example: The breast-fed baby at a given age who is accustomed to a certain quantity of breast milk, may, during dentition, feel nature's influence and because of impaired appetite incident to diminished power of digestion take only one-half or one-fourth of the accustomed quantity of breast milk; unless the feeding is weighed, no one is the wiser. On the other hand, a bottle-fed baby of the same age and accustomed to the same quantity of food, who takes only one-half or one-fourth of the usual quantity because of the same factor, immediately excites the concern of the parent. In many instances, the food is changed for another combination, with which the baby has had no experience and his digestion, already diminished, is further impaired and a disturbance produced. A diminution in the strength of the original food and an increase of the water content would have averted the trouble, and the early return of the baby's appetite would have indicated its preparedness for increase in the food allowance with gradual approach to the original mixture.

The appetite in disease is a good index for dietetic treatment, while vomiting is nature's safety valve against an over-supply or improper food. During disease, when the appetite is absent, the body draws upon its adipose tissue, which rapidly becomes exhausted, and also upon its water content, so that a process of drying out takes place. The fluid content may be easily replaced, for thirst is almost always present and water may be given freely except in rare instances in which there is already a storage rather than a loss of tissue fluids. The free administration of water serves to flush out waste material, which is of decided value especially when toxins are present, the elimination of which is thereby facilitated with the least irritation. Of the food elements in febrile conditions fats are not well tolerated and frequently increase the tendency to gastric symptoms. Proteins should also be diminished to a minimum. Carbohydrates, both soluble and insoluble, are indicated as their functions in nutrition are exercised to their fullest capacity. It is rarely necessary to give much consideration to the salts. There are, however, conditions in which these may be injurious, when due attention must be given to reducing this injury to the smallest possible degree.

The age of the patient and the character of the illness have important bearing upon the dietetic management in disease. A thorough knowledge of digestion in the infant, and of the individual capacity is of decided advantage, especially during the first and second years of life. The character of the disease, that is, whether it is self-limited or chronic, will have some bearing on the administration of food. In the former, aside from attempting to spare the body waste, carbohydrates (preferably soluble) tend to lessen the severity of the disease by diminishing the possibility of a complicating acidosis and thereby to conserve the strength and promote the resistance of the individual. The reappearance of the appetite may be regarded usually as an indication to increase the diet.

In chronic diseases it becomes essential to feed the patient as well as possible so as to break the vicious circle into which such a patient usually lapses with the view to promote the appetite by increasing the individual's resistance and strength.

The food requirement in illness, it should always be remembered, is less than in health. While the patient is recumbent and does not exercise the coloric demand is reduced to a nimimum. Care should, therefore, be exercised lest an upset be produced by overfeeding.

As has already been stated the appetite is a good index for the increase of food. However, if elimination is not active, the return of the appetite may be unduly retarded. This is especially true if constipation exists. It is essential that there should be a daily evacuation of the bowels during illness unless there is some contraindication, as acute appendicitis.

Certain diseases require special dietetic treatment, as disorders of the gastro-intestinal tract. In passing, it should be remarked that it is wiser to prevent digestive disturbances than to cure them. By careful management and feeding in many diseases, particularly those which predispose to gastro-intestinal complications, much trouble may be averted. The diseases requiring special dietetic measures will be considered elsewhere.

In order to obtain the greatest benefit from the dietetic management of sick children certain fundamental principles must be respected. In a normal healthy individual with a digestive apparatus of maximum efficiency digestion may easily be disturbed, particularly by irregularity in meals. In a sick person with diminished and deficient digestion, the effect of such irregularity will be more pronounced. The digestive system of the small child and infant is very delicate and consequently any indiscretion will lead to more severe results. Hence, the necessity of regularity in the feeding of sick children. Not only should this regularity apply to intervals between meals, but also to the hour of the meals.

It is at times very difficult to carry out the dietetic management particularly when dealing with capricious children. In these instances disciplinary methods become imperative. A well governed child is much more easily managed during illness and the chances for recovery are greater than in the child which is humored and pampered by indulgent parents.

PSYCHOTHERAPY

Who has not witnessed with increasing interest the unfolding of the infant mind? With a thorough understanding of the nutrition and development of the brain proper steps can be taken to assure a firm foundation for a stable nervous system. A child with nervous balance can be more easily controlled in health, while in serious illness its ready coöperation will favorably influence the outcome of the disease.

Psychotherapy, or suggestive therapy, may be applied by varying methods in accordance with the requirements of an individual case. Of prime importance are the coöperation of the parent or guardian and harmonious surroundings, otherwise the attempt may be unsuccessful or results are realized with difficulty. The outcome of any psychotherapeutic measure will in great part be influenced by the disposition of the patient toward the physican at the first visit, and the results will depend upon the physician gaining the confidence and appreciation of the patient in every treatment. A negative or pessimistic frame of mind will handicap the situation and the outcome will be problematic. However, the maturer mind may by persistence gain supremacy over the weaker one, particularly when the influence is exerted with kind indifference.

A thorough knowledge of the inner life of the child must be appreciated; its toys and plays, its likes and dislikes must be respected. It must not be antagonized nor deceived. If a child is to experience pain, it should not be misled but apologies should be offered beforehand and the impression given that the discomfort or pain is a necessary incident in the process of getting well. The child should be encouraged to lend confidence not only to its attendant but to itself.

There should be no suggestion, as is usually the case, of fear, in enforcing compliance with the physician's directions, nor should the physician be used as a threat when desiring obedience. Rewards and deprivations serve to better purpose than threats. In short, the little individual should be treated in a friendly and tactful manner.

Ease of accomplishment in this will depend in great measure upon hereditary and environmental influences. It is practically the only way in which preventive measures are more effective than corrective measures. Home influence of parents or grandparents or other members of the family are at times very harmful, especially in neurotic diseases. Familial tendencies coupled with the aforesaid contributing factors are productive of marked cases of hysterical symptoms in childhood, as well as of milder nervous disturbances. It may become necessary, in order to break the vicious circle into which some cases progress either consciously or unconsciously, to place a child in the care of strangers who have better control over it than the mother, and in extreme cases a complete change of surroundings may be necessary.

Success in psychotherapy will in great measure depend upon the thoroughness of the examination and upon the physician's knowledge of the psychic influence in all diseases. The promptness with which psychotherapy is employed may influence the duration and severity of many nervous diseases, namely, by preventing the fixation of diseased conceptions. Greater familiarity with the advantages of this therapeutic aid will extend the field for its employment.

As a rule verbal suggestion will suffice, but, in some cases there will be found an unfavorable habit, inherited tendency, or frame of mind, that influences the disposition of the patient. In such cases suggestive therapeutics may be assisted by drugs, massage, regulation of diet, rest, hydrotherapy, electricity, exercise, passive or active sleep.

The character of the growing healthy child may be properly molded by the right surroundings, surveillance, and regulation of routine. Special care should be given to rest, sleep, nourishment, outing, association with other children of the same age, play, education and habits.

THERAPEUTIC TECHNIC

While physicotherapy in its strictest sense implies the treatment of disease by natural agents, such as light, air, electricity, it will be considered here in a modified sense, including certain physical procedures related to therapeutic technique. The physicotherapeutic measures most frequently employed in infancy and childhood are directed to the treatment of the digestive tract.

Gastric Lavage.—Washing the stomach is one of the most valuable therapeutic procedures at our command. It is based on the introduction of water into the stomach and its withdrawal by siphoning. The apparatus consists of a soft rubber catheter, the caliber depending on the size of the patient (usually No. 12 or 16 American with one large eye near the end), a piece of glass tubing 2 in. long to connect the catheter to a ¼-in. rubber tubing about 2 ft. long, and a small glass funnel of 4 to 6 oz. capacity attached to the other end of the rubber tubing. Also necessary are a pitcher, basin and rubber sheet.

For gastric lavage the patient, with his arms at his sides, is snugly enveloped in a rubber sheet and held, preferably seated in the lap of an assistant, his legs fixed securely between the knees of the assistant, his head steadied by the left hand, and his body by the right arm of the assistant. The patient may also be laid on his back. No mouth gag is necessary with infants but with older children one should be used. The type selected should be well protected by pieces of rubber so as to prevent laceration of the gums. The catheter, previously boiled, is introduced by the physician whose left forefinger serves as a tongue depressor and guide, the right hand passing the catheter rapidly over the tongue through the pharynx and into the esophagus. The catheter should be moistened with the fluid to be employed and not with vaseline. As the tube passes the pharynx the child will gag, retch, and may become flushed. This should cause no alarm, since it is exceedingly difficult for the tube to pass anywhere except down the

Note.—Many of the remedial measures outlined under this chapter are not "physicotherapy." "Special Measures" or "Therapeutic Technic" would possibly be more appropriate.

pharynx. Presently the child will cry and begin to breathe regularly. The tube should be passed rapidly through the mouth and pharynx because, if the patient gags and resists at this stage, the procedure is rendered more difficult. When the tube is in the esophagus it may be passed more slowly. The catheter should be passed a little beyond the cardiac orifice of the stomach and when it reaches this point there may be a second temporary gagging. The distance from the teeth or the gums to the stomach will vary according to the size of the child, usually about 10 in. and rarely less than 9 in. However, the entrance of the tube into the stomach is easily recognized by the distinct reflex at the cardiac orifice, and often by the escape of curdled milk or stomach contents into the tube and funnel. When the catheter has entered the stomach the funnel should be raised high to allow the gas to escape, and then, should gastric contents come up into the funnel, the latter may be immediately lowered so as to siphon away as much as possible into a basin. The funnel should again be raised and water introduced. At times for a temporary period the water will remain stationary but this will be promptly followed by a free flow.

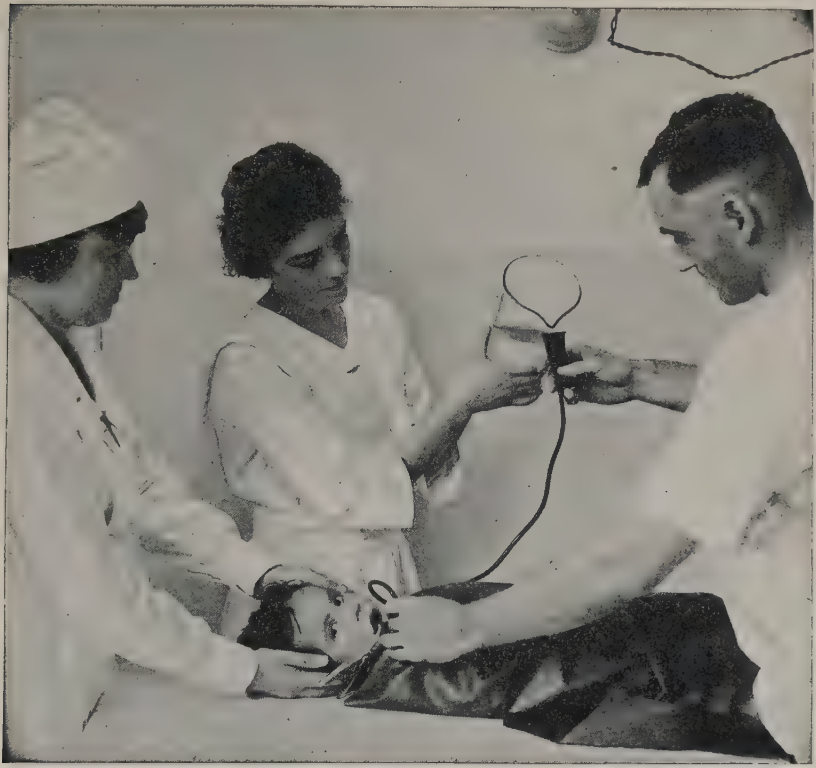

FIG. 106.—Gastric lavage.

Before the funnel is entirely empty it should be quickly lowered so as to create a siphon action. The amount of water should be approximately the amount of feeding at the age of the child. The temperature of the water should be from 100 to 110°F., and the water should be introduced slowly. The higher temperatures are indicated if gastric irritation is marked. It may be necessary to use quite a quantity of water, particularly if the curds of the stomach interfere with the flow as they are gradually broken up. If too much water is used the patient will vomit. As soon as the child shows discomfort the funnel should be immediately lowered as this may be an indication of gastric over-distention. If the return shows mucus, sodium bicarbonate should be added, a teaspoonful to the pint of water. When there is much thirst a little water may be allowed to remain in the stomach.

In withdrawing the catheter, should it be desired to allow the water or any material to remain in the stomach, the tube should be pressed so as not to permit any dripping while the catheter end passes through the pharynx, as this would stimulate vomiting.

Lavage is employed with more facility in infants, i.e., during the first two years of life. In children about three years old, greater difficulty is encountered, owing to the alarm and consequent struggles of the child; but at this time of life the procedure may be carried out through the nose, necessarily with a modified apparatus.

Lavage is employed to (1) remove poisons and introduce antidotes; (2) to prevent irritation and gastric dilatation in pyloric obstruction; (3) to prevent distention from food changes, and to relieve discomfort in gastric dilatation; (4) to clear the stomach in acute gastric indigestion, and (5) to remove mucus in chronic gastric indigestion.

Gavage signifies enforced feeding and consists in introducing food through a tube into the stomach. The apparatus employed is the same as for gastric lavage. The technique is identical except that it is preferable to have the child in a recumbent position, and the patient should not be disturbed for a period following the procedure. After the tube has been introduced into the stomach the funnel is elevated to permit of the escape of gas, the stomach is emptied of its contents and washed, and then the desired food is introduced. In removing the catheter after all the food is believed to have reached the stomach the tube is pinched so as to avoid any trickling into the pharynx, and the tube is withdrawn quickly. Should the food be vomited more may be introduced at once.

Gavage is indicated: (1) In premature and asthenic infants who are otherwise unable to take food; (2) in cases of habitual or persistent vomiting where swallowed food is frequently regurgitated, whereas food given by tube is retained; (3) occasionally following intubation or certain operations on the mouth and esophagus; (4) in many acute diseases, particularly in septic cases in which the child refuses to take any or an adequate food, as in diphtheria, scarlet fever, typhoid fever; (5) in cases of multiple neuritis after diphtheria with marked involvement of the muscles of deglutition, of the palate, pharynx and larynx, with a tendency to aspirate food, and (6) in cerebral disease when food is not taken because of delirium or coma.

The foods given by gavage may be breast milk, full strength or diluted, modified or peptonized cow's milk, plain or dextrinized gruels as indicated, because in cases in which this method is resorted to the digestion is usually feeble. The amount at each feeding should be below the usual requirements or the intervals of feeding lengthened, the food, of course, being sufficiently warmed. Stimulants and medicine well diluted may sometimes be administered in this way.

The procedure is especially indicated in infancy, as in older children there may be considerable difficulty, necessitating the use of a mouth gag and assistants, in which instances nasal feeding would be more easily accomplished.

In **nasal feeding** the food enters the stomach by means of a tube passed through the nose. The method is similar to gavage but the apparatus is modified by employing a No. 10 American catheter. The indications are the same as for gavage, but this method is preferable in children over two years of age in whom the tube cannot be introduced through the mouth, or only with a great amount of struggling. As the tube is much smaller than for lavage and gavage, care should be exercised not to enter the larynx, not a probable but a possible accident. No food should be introduced until it has been established with certainty that the larynx has not been entered. Aphonia and a hissing sound will indicate that the tube has entered the larynx.

Breck Feeding-tube.—The feeding of premature infants is at times made difficult by the inability of the infant to grasp the breast or to suck. In some instances feeding may be successfully carried out by using a dropper, pipet, or a nursing tube constructed for this purpose.

Enema.—An enema is an injection through the anus into the bowel, either the rectum or higher up. It may be employed for the purpose of (1) causing a stool; (2) softening the contents in the rectum to overcome constipation when due to hard, dry stools; (3) introducing nourishment and (4) introducing medicines.

Enemas per se are the greatest contributors to the production of chronic constipation. The results attained may be spectacular, but if the measure is resorted to injudiciously the after effect may require patience and perseverence to overcome. At times, however, when it is necessary to produce a prompt evacuation of the bowel, as for instance in colic, an enema of a teaspoonful of glycerin to an ounce of water will usually give immediate results in an infant, a larger quantity being required for older children. An ordinary soap-suds enema may also be employed with satisfactory results. It is surprising, however, how often a plain warm water enema will produce the desired effect. In giving enemas for the evacuation of the bowel a bulb syringe, 4 oz. capacity, with a hard rubber nozzle, or preferably an "eye or ulcer" syringe of the same capacity with a soft tip will suffice, the tip being lubricated before introduction. The contents of the syringe is expressed after the tip has passed beyond the anus, the child lying on its back.

In constipation when the fecal mass is hard and dry an enema of olive oil, ½ to 1 oz., may be used. This may be followed with a soap-suds enema if the oil enema does not suffice. When the stools are chronically hard, which occurs often as a result of too many stimulating enemas and because the fecal mass remains in the rectum too long, an enema of olive oil, about 2 oz., may be given at night. This is not intended to produce an immediate result but to be retained with a view to lubricating the parts and softening the hard fecal masses. This condition is brought on by the lack of inclination to stool owing to the insensitivity of the bowel to the natural influences because of the excessive stimulation induced by the irritating enemas.

For oil enemas a bulb syringe with a hard rubber nozzle is preferable. A small rectal tube or a No. 18 American catheter about 9 in. long is attached to the hard rubber nozzle. The tube should be well lubricated, and while the child is in the dorsal position, or better still in the Sims position with his hips elevated, the tube is passed carefully for its full length. Then the contents of the syringe are emptied into the bowel. The oil should be introduced high into the bowel if it is desired that it be retained during the night, and the enema should not be given until the child is in bed for the night.

Nutrient enemas have a limited application in infancy and childhood (1) because the rectum soon becomes intolerant and (2) because of the questionable efficacy of the various nutrients used by this route. Peptonized milk was formerly employed rather generally and still has its advocates. Glucose, however, is the most beneficial available nutrient for this purpose; it should be given in 6 per cent. solution and in varying quantities dependent upon the age of the child. Nutrient enemas should be given at intervals of not less than four hours and in small quantities. A young infant will rarely tolerate more than 1 or 2 oz.; a child at two years 2 to 4 oz. Older children may retain a greater quantity. Nutrient enemas are always given with the view to their retention and are employed for giving temporary relief to the stomach.

Medicated enemas may prove useful for the administration of drugs that have an unpleasant taste or cause vomiting. The quantity should be the same as for the nutrient enemas, and the drug administered either in water or gruel; the latter will probably be better tolerated. Drugs such as quinin and chloral, particularly the latter, are advantageously administered in this manner.

The temperature of the enemas to be retained should be about 100°F. and not less than 95°F. Enemas given too hot or too cold will not be retained. When giving nutrient and medicated enemas, after the withdrawal of the tube the buttocks should be pressed together for a half hour to prevent expulsion.

Before giving nutrient and medicated enemas the bowel should first be cleansed by flushing unless it is previously empty, and preferably about half an hour later the enema should be given using a No. 18 American catheter or a small rectal tube attached to the small tip of a fountain syringe. The child should be placed on its left side or in the Sims position with the buttocks elevated by means of a pillow to from 4 to 6 in. above the level of the shoulders. The tube and the anus should be well oiled. The tube is then passed into the bowel for about 9 in. before the contents are allowed to flow from the syringe, which should be from 2 to 3 ft. above the patient. The tube is then rapidly withdrawn and the patient kept in the same position for about one-half hour. In older children the piston syringe or a Davidson syringe may be required to overcome the pressure exerted by the patient.

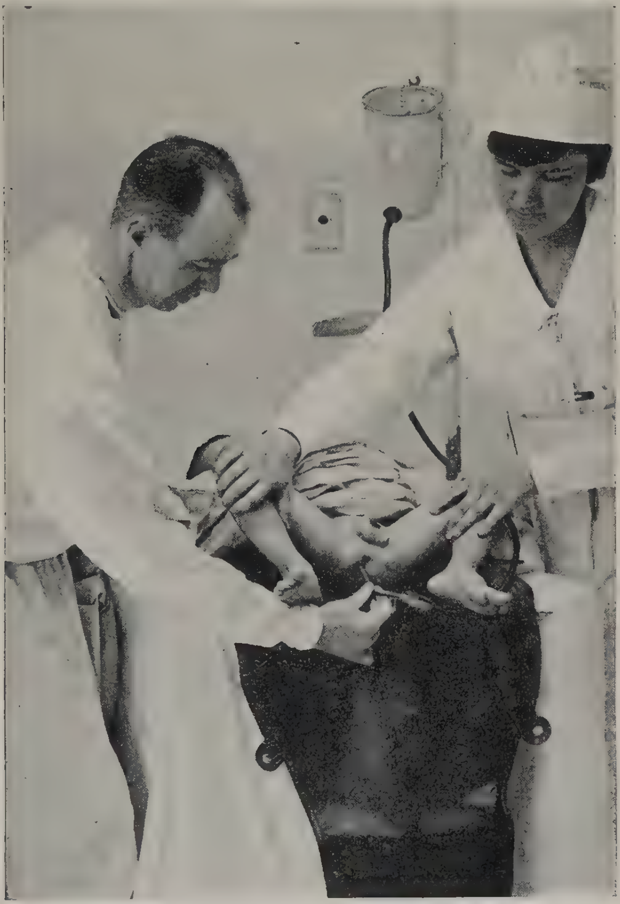

FIG. 107.—Irrigation of the colon.

Colonic irrigation signifies the flushing of the large intestine with large quantities of water introduced as high up as possible by means of a soft rectal tube or catheter (Fig. 2).

The apparatus consists of an irrigating can or ordinary fountain syringe, 4 or 5 ft. of rubber tubing, and a soft rectal tube or No. 18 American soft rubber catheter. The catheter should be attached to the nozzle of the fountain syringe. An irrigating can or a pitcher and funnel may also be used. The double current Kemp tube or two catheters of different sizes (the larger for the outflow and the smaller for the inflow) may be employed with advantage in cases in which the prostration is so great as to cause entire loss of muscle tone. In these cases the capacity of the colon should be known. At six months of age the colon without distention has a capacity of one pint, and at two years from two to three pints. However, even in these cases the single catheter will suffice if judiciously used with the fountain syringe at a low height, the tube being disconnected from time to time to allow the fluid to escape and to avoid any injury from pressure. The child should be placed on its back with the thighs flexed upon the trunk, the buttocks brought to the edge of the table or bed, and either a Kelly pad or rubber sheeting arranged in such a manner as to act as a trough that empties into a basin on the floor. When it is desired that the fluid shall be retained the patient is placed on the left side with the thighs flexed on the trunk and the hips elevated. The fountain syringe should be from 2 to 3 ft. above the patient. The catheter or rectal tube should be greased with vaseline or olive oil, the flow turned on so as to allow the escape of the cold water, and the tube introduced slowly with the water running. The running water distends the bowel ahead of the tube and facilitates its passage. Occasionally the catheter may become turned upon itself; it should then be withdrawn and reintroduced. If the patient resists the procedure strenuously, advantage may be taken of the breathing movements to introduce the tube by pushing it forward at inspiration. The tube should be introduced from 9 to 14 in. depending upon the size of the patient. By palpating the left side over the colon it may be ascertained if the tube has been satisfactorily introduced. No concern should be felt, except in the cases already mentioned, about the return flow, which will be expelled beside the catheter when the patient forces it out. The tube should be held particularly close to the buttocks at this time as otherwise it may be expelled. The flushing should continue until the return flow is clear. Irrigations of less than a gallon are usually valueless. From a pint to a quart can be introduced before the patient endeavors to pass it. Normal saline solution, a teaspoonful of common salt to the pint of water, is ordinarily used at a temperature of from 95 to 100°F. A change in the position of the child and gentle massage of the abdomen will facilitate the introduction of the fluid into the upper part of the colon. This is, however, not absolutely necessary. Irrigation of the colon always stimulates peristalsis of the lower ileum so that this segment is usually emptied during this procedure. When the fluid returns clear and the flushing is complete the tube may be withdrawn without disconnecting it from the nozzle of the fountain syringe, or it may be disconnected and the fluid allowed to escape from the bowel. Some of the fluid always remains—a pint or more—which will be passed in about half an hour or so. If desired a medicament can be introduced high up into the bowel after the irrigation; the tube is withdrawn after sufficient time has elasped for the medicament to escape from the tube.

The indications for colonic irrigation are: (1) To cleanse the bowel of undigested food, decomposing secretions, fecal material and mucus, for which normal saline solution is used, or in the case of removing mucus, a solution of bicarbonate of soda ½ oz. to a pint of water, may be of advantage; (2) as a means of introducing medication in inflammatory conditions of the colon; (3) to reduce temperature, the double flow or the single tube being employed, disconnecting the latter as frequently as necessary to permit the outflow of the warmed water, the temperature of the water being about 70°F.; (4) as a means of relief when there is much pain, tenseness and straining, the water being employed as in the foregoing at 90°F., gradually reduced to 70°F.; (5) for collapse with subnormal temperature, a hot saline solution at 105 to 110°F. having a stimulating effect; and (6) as a means of stimulating kidney function, allowing as much of the fluid to be retained as possible.

While colonic irrigation when indicated is a definite and valuable therapeutic agent in the treatment of diseases of children, unfortunately it is too frequently employed injudiciously, with discouraging results.

Murphy Drip.—One of the most valuable therapeutic measures for the introduction of fluids into the body by way of the bowel is by the drop method (Fig. 108). The apparatus comprises a thermos bottle with a rubber stopper through which are passed two glass tubes, the one extending to the bottom of the bottle and the other just within the stopper. The bottle is suspended upside down. A rubber tube 5 or 6 ft. long is cut into two pieces; one of these is attached at one end to the shorter glass tube that extends just within the bottle and at its distal end to a glass tube (Murphy drip tube). The other piece of tubing is attached to the distal end of this glass (M.D.) tube and at its distal end by means of a glass connection to a small catheter. The thermos bottle is generally used for the purpose of keeping the water hot, but since the water must course for a considerable distance and is dropped slowly, it is quite cool before it reaches the catheter, hence, this advantage of the thermos bottle is lost. A fountain syringe bag will therefore serve equally well as a container. The container should be suspended at a convenient height. The catheter is introduced well into the bowel for about 6 or 8 in. The rate of flow is regulated by means of a screw compressor above the Murphy drip glass tube, and hot-water bags are applied around the tubes as near to the anal entry as is possible without injuring the patient. This serves to warm the water before it enters the body. The rate of flow should be begun at about 10 drops a minute and may be increased to 15 or

18 drops per minute. Any attempt to allow a more rapid flow will meet with unsatisfactory results. The fluid may be given continuously, but in younger patients it should be given preferably for an hour at a time at intervals of an hour. By the latter method the drip may be employed over a longer period before the subject becomes intolerant and a larger quantity of fluid can be introduced during twenty-four hours. The Murphy drip is indicated: (1) In cases in which the stomach is extremely irritable; (2) in cases of incessant and uncontrollable vomiting; (3) in cases in which there is great loss of body fluid, and (4) as a means of introducing nutriment (glucose), saline solutions, and medication.

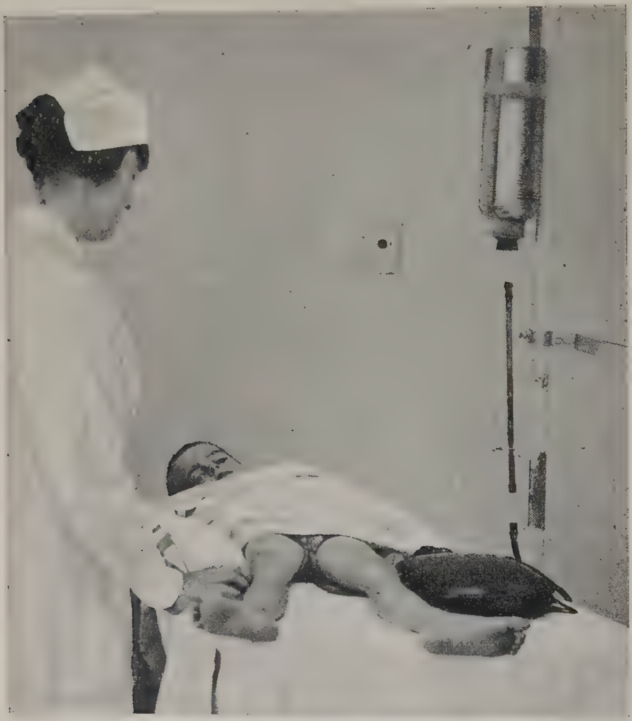

FIG. 108.—Introducing fluid into the rectum by means of the Murphy drip.

Suppositories are usually employed too indiscriminately in childhood, infancy, and more particularly in early infancy. They are probably the greatest contributors to chronic constipation, but when employed judiciously may be valuable agents for producing prompt evacuation and for overcoming constipation. They may also serve for administering drugs. They should never be used as a routine measure because the bowel readily becomes dependent upon their excessive stimulation, becomes insensible to the natural stimulation, and may later fail to act.

Suppositories should be employed only as a temporary measure and are most useful when the constipation is due to rectal causes. They may be of assistance in establishing regularity of the bowel movement by training the infant to have a stool at the same hour every day. The coned oil paper may be of assistance in forming the proper habit.

Suppositories of glutin, soap and glycerin may also be employed. The glutin suppositories are less objectionable than the other two in that they are less irritating. They are, however, slower in action. Soap suppositories cause an immediate effect owing to their marked irritative power, but should be used with caution or not at all as the same results may be produced by less injurious methods. Glycerin suppositories are even more objectionable than those of soap and while convenient, their frequent use may cause much irritation and even proctitis.

Unfortunately, suppositories are too frequently employed by lazy caretakers as a means of obtaining a prompt result with the least effort and without regard for the future comfort, well being, and habits of the defenseless child.

Nasal Spray.—An ordinary hand atomizer is employed for spraying the nose, the solutions used being either aqueous or oily. Of the oily solutions liquid petrolatum or benzoinol are perhaps the most satisfactory and are of especial advantage when the nasal mucous membrane is dry. The aqueous sprays, usually an antiseptic or alkaline solution, are employed for cleansing purposes. Those most frequently used are boric acid in 2 per cent. solution, Dobell's solution, or Seiler's solution, or a normal sodium chloride solution.

Nasal Irrigation.—Nasal irrigation is indicated when it is desired to cleanse the nose and nasopharynx. The apparatus consists in either a piston syringe, when a small amount of fluid is to be used, or an ordinary fountain syringe for larger amounts. The fountain syringe gives a uniform flow that is easily regulated by altering its height above the patient, which should rarely exceed 2 ft. If a fountain syringe is used the child's mouth should be kept open to lessen the likelihood of forcing the water into the Eustachian tubes.

A piston syringe should have a soft rubber tip sufficiently large to fill the nostril so that no injury may be done to the nasal mucous membrane. Before irrigating the nasal cavity the apparatus must be thoroughly sterilized, as otherwise it may be the means of disseminating disease.

For irrigating the nose the patient may be either in the recumbent or sitting position. If the infant is too weak, or in diphtheria, scarlet fever, or conditions of depression, the procedure may be carried out without removing the patient from the bed. The head is turned to one side with a pus basin beneath the nose. The patient may be enveloped in a sheet so that he will not employ his hands to interfere with the procedure.

The solution is allowed to flow through the upper nostril until it returns through the other nostril or through the mouth, the latter indicating that the rhinopharynx has been reached. After one side has been thoroughly irrigated the head of the patient is turned to the other side and the procedure is repeated. The syringe should be held on a plane parallel to the floor of the nares.

In the sitting position the patient, enveloped as before, is held by an attendant with the head inclined a little forward and the mouth opened; the syringe is held parallel to the floor of the nares and the fluid is allowed to flow into the nose and return through the mouth or the other nostril. Neither method causes any depression or prostration.

Gargles.—Gargling can only be employed in older children. It is useful in many diseases of the mouth and pharnyx, the character of the solution depending upon the pathologic condition in which it is indicated.

Syringing the Mouth and Pharynx.—In those children unable to gargle, syringing of the mouth and throat may be carried out when indicated. The child may lie upon its side or be held in the sitting position with its head inclined forward. Either a piston syringe, bulb syringe, or fountain syringe may be used. The nozzle is placed at the angle of the mouth between the back teeth and by acting as a tongue depressor the pharynx may be easily reached.

Nasal packing is employed in uncontrollable epistaxis. It consists in plugging the anterior and posterior nares. A small catheter is passed through the anterior nares and is caught in the pharynx. A piece of heavy thread, to which is fastened a piece of gauze at one end, is attached to the oral end of the catheter. The catheter is then withdrawn by way of the nose until the gauze plug closes the posterior nares. The gauze plug should be the size of the terminal phalanx of the thumb of the patient. If the gauze is saturated in tincture of benzoin it has a more soothing and beneficial effect. This packing should not be allowed to remain in longer than 24 hours, when it should either be removed or changed. In this way damage to the Eustachian tube will be averted. The anterior nares are plugged by strips of gauze inserted along the floor of the nasal cavity until the cavity is entirely plugged.

Inhalations are useful in many of the affections of the respiratory tract. They may consist of either steam or vapor that is medicated by adding some drug to the boiling water. To be efficient the crib or bed should be entirely enclosed by sheets, forming a tent. Various types of apparatus may be employed, namely, the croup kettle, vaporizer, or steam atomizer. The alcohol lamp, which is attended with much danger, should whenever possible be replaced by an electric heater. The generator, whether it be a croup kettle or vaporizer, should be placed at a safe distance from the tent and some suitable means employed to conduct the steam into the tent. There are various special apparatuses for administering medicated vapors. The ordinary croup kettle usually suffices for medicated steam inhalations, the drug being added to the boiling water in the kettle. Some croup kettles have a receptacle in the nozzle or spout for a sponge which is saturated with the medicament, the vapor passes through the sponge and becomes impregnated with the medicament.

Calomel inhalation may be used in croup, more particularly in membranous laryngitis due to streptococcus infection, or in diphtheric laryngitis when antitoxin is not available. About 10 gr. of calomel is placed in a tin spoon or other receptable over a flame and vaporized, the fumes being led under the tent the air of which has first been moistened with steam vapor. In administering calomel inhalations the possibility of salivation must be borne in mind.

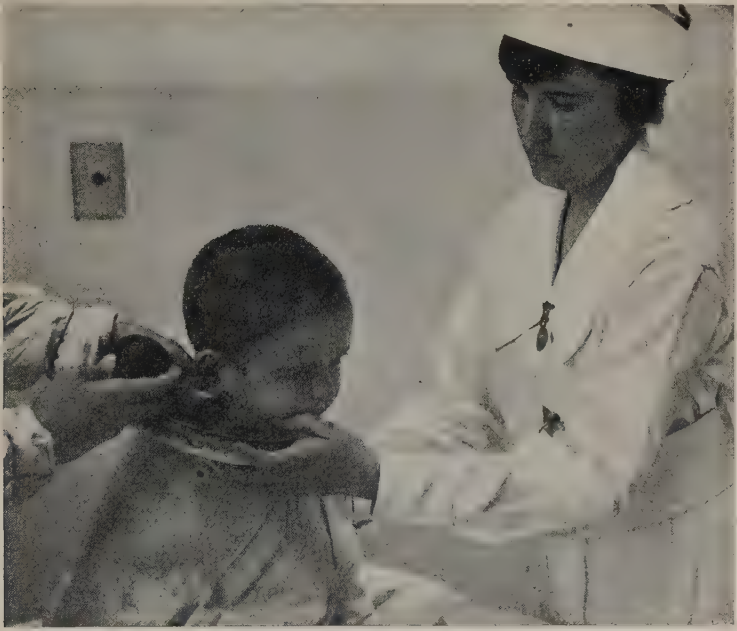

FIG. 109.—Irrigation of the ear.

Inhalations may be employed directly without apparatus, such drugs as ammonia, amyl nitrite and chloroform being administered by inhalation.

The oiled silk jacket is employed in respiratory affections for maintaining a uniform temperature of the thorax and for continued counterirritation of the thorax, the jacket necessitating less frequent application of the counterirritant. The oiled silk should be lined with flannelet or a woolen material, and the jacket should cover the entire thorax and extend over the shoulders, leaving only room for the arms, it should close on the side and reach below the crest of the pelvic bones.

Intubation.—This therapeutic measure, employed to relieve laryngeal stenosis, will be considered in the discussion of diphtheria, in which it is more specifically indicated.

Syringing of Ears.—For syringing the ears a bulb ear-syringe of soft rubber or a fountain syringe may be used. The patient may be seated or in the recumbent position with a basin beneath the ear to receive the return flow (Fig. 109). The lobe of the ear is slightly pulled out and down from the head so as to straighten the auditory canal. The nozzle of the syringe is then introduced into the external auditory canal, care being taken that the syringe does not occlude the opening of the canal and thus interfere with the return flow along the nozzle. A saline solution is ordinarily employed. Owing to the frequency of middle ear disease in early life this therapeutic measure is quite commonly employed.

Spinal or lumbar puncture consists in tapping the subarachnoid space usually between the third and fourth or the fourth and fifth lumbar vertebræ at a point along an imaginary line joining the highest parts of the iliac crests (Fig. 110). The procedure is more of diagnostic than therapeutic importance though specific sera as well as drugs are introduced in this manner. It also affords a means of relieving pressure symptoms of the nervous system. Special consideration is given to this subject elsewhere.

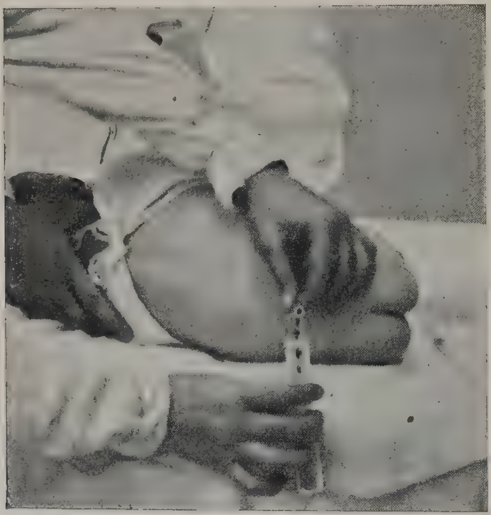

FIG. 110.—Position of the child in spinal puncture.

Lateral ventricle tapping is employed for the same purpose as lumbar puncture, being indicated when no fluid is obtained by lumbar puncture in cases with evidences of intracranial pressure, successful lumbar puncture being prevented by closure of the foramen of Magendie. It is best performed on infants with open fontanels, the needle being introduced to a depth of at least an inch at the lateral angle of the anterior fontanel downward and inward into the lateral ventricle. Naturally, this should be done under aseptic precautions.

Paracentesis of thorax and pericardium serves particularly as a diagnostic measure. However, if there are very large serous effusions aspiration may relieve distressing symptoms and may result in rapid absorption. The technique is described in connection with the specific subject.

Paracentesis of the tympanic membrane is an important procedure in infancy and childhood because of the ease with which infections from the nose and throat extend to the middle ear through the Eustachian tubes. This extension is especially frequent in children who do not blow their noses or who do so incorrectly. Paracentesis can be performed very quickly and, when acute respiratory diseases are present, preferably without a general anesthetic because of the likelihood of extension of the infection into the lung through irritation of the mucous membrane. However, local anesthetics may be applied to the drum membrane (as, for example: Carbolic acid) to lessen the pain. When it is not contraindicated ethyl chlorid may be administered for general anesthesia. Paracentesis is attended with very little risk and should be done under the most cleanly conditions. When done under unclean conditions there is danger of transforming a simple into an infectious purulent inflammation of the middle ear.

The course of a simple otitis media may be distinctly shortened by paracentesis, which allows the serous and serohemorrhagic exudate to escape and serves to relieve pressure within the ear and to lessen resorption. On the other hand, procrastination may lead to irremediable damage which may result in severe and even fatal complications.

Cutaneous Therapy.—Simple massage of the skin, particularly after a bath, will promote the circulation and nutrition. The skin should be thoroughly rubbed until dry. Olive oil and cod-liver oil may be rubbed into the skin, as may also certain drugs such as mercury, quinin, etc. The effect of mercury administered in this way is quite pronounced, quinin, however, is absorbed only in small quantities. Other drugs that are readily absorbed by the skin are carbolic acid and salicylic acid and their congeners. Caution should be exercised in the employment of these drugs, particularly in infancy.

Counterirritation.—Counterirritation is employed in many diseases of infancy and childhood, but severe methods of blistering are almost never indicated. Counterirritation may be used for a transitory effect or for a prolonged period, over a circumscribed area or large surface. The desired effect will influence the choice of method. The simple measures will ordinarily suffice to produce reddening of the skin. It must be remembered, however, that the skin is very delicate and the agent therefore must be employed with caution.

Mustard poultice is made of one part of mustard and six parts of flax-seed or flour. The mustard is rubbed up in cold water until free from lumps; it is added to the boiled meal and thoroughly mixed with it just before use. The mixture is spread over a double layer of cheese cloth, the latter is folded over and the poultice is applied. For local counterirritation the poultice may be used satisfactorily, but in pulmonary disease the practice of enveloping the entire thorax in a mustard poultice must be condemned as it causes restriction of respiratory movements. This may seriously handicap the very young infant, particularly when the respiration is already difficult. The poultice should be applied warm, its effect on the skin carefully watched, and it should be removed when redness of the skin has been produced, which should be within 5 or 10 minutes. If the poultice remains in place too long it will become cold and clammy and have an effect opposite to that desired. The poultice may be applied every three hours for several days if it does not injure the skin.

Mustard Pack.—Mustard in the proportion of one tablespoonful to the gallon of water is employed for this purpose. A sheet or towel saturated with the tepid mustard water is wrapped around the naked child and over this a blanket is rolled. The patient should remain in the pack until the cutaneous effect is produced, usually 10 to 15 minutes. The pack may be repeated when indicated, within the hour if necessary. While the mustard pack is not quite as efficient as the mustard bath it has the advantage of causing less disturbance to the patient. It is useful in conditions of collapse, prostration, cerebral and pulmonary congestion, and convulsions.

The Turpentine Stupe.—This is made by immersing a piece of flannel in water as hot as can be borne by the hand. The flannel is wrung dry, and from 10 to 15 drops of turpentine are sprinkled upon it, after which it is applied to the surface of the skin, and covered with dry flannel or oiled silk. Care should be exercised to avoid blistering of the skin. Turpentine stupes are indicated in abdominal distention and abdominal pain or inflammation.

Turpentine and Oil.—This method of counterirritation is probably the best means of obtaining continuous uniform counterirritation of the skin and when properly employed does not cause blistering and injury of the skin. It consists in applying to the surface a mixture of turpentine, eight parts, and olive oil, one part, covered with flannel and oiled silk. The effect on the skin should be watched. Should the skin become unduly red, an application of olive oil will prevent further reddening. In the stated strength the redness is rarely intense, in fact it is frequently necessary to increase the turpentine in the mixture even up to 12 parts to 1 of olive oil. The method is specially indicated in pulmonary disease, particularly in pneumonias where it may be employed continuously without harmful effect on the skin or kidneys (by absorption). It has the decided advantage of producing counterirritation of the entire thoracic surface without restricting the respiratory movements of the child or small infant. It may be employed as often as required, even every hour if necessary. The effect may be prolonged if an oiled silk jacket lined with flannel is worn.

Dry cupping may serve in childhood and even in infancy as a useful means of counterirritation to relieve the acute congestion of pneumonias, bronchitis or pulmonary edema with marked dyspnea and cyanosis, also in mastoid disease, and at times in nephritis. Dry cupping may be performed with cupping glasses and a suction pump, the glasses being held firmly in position and the air exhausted. Small tumblers or wine-glasses will answer the purpose, however. In the use of the latter, their interiors are moistened with a little alcohol, this is lighted and the cup is immediately applied and held firmly over the part. As the heated air cools the resulting contraction will produce suction. For removing the cups air should be allowed to enter either by opening the stop-cock of the cupping glass, or by tilting the wine-glass so that its rim loses contact with the skin at one point. A modified form of dry cupping consists of the application of Bier's suction cups, which are attached to rubber balls by means of which suction can be exerted, producing passive hyperemia. This method is useful particularly in the treatment of boils, carbuncles and enlarged glands.

Local blood letting is employed as a measure of relief in the early stages of inflammation of the mastoid or middle ear. Leeches may be used for this purpose. Wet cupping is never indicated in young children.

Bleeding.—General bleeding is seldom indicated in early life. The withdrawal of from 3 to 4 oz. of blood is advocated in the severe cyanosis of pneumonia from dilatation of the right heart, and in uremia. A constrictor or a wet gauze bandage is applied around the pendant arm until the veins become distended. A small incision is then made into the vein at the bend of the elbow in the direction of the course of the vessel. After the desired quantity of blood has been allowed to flow, the bandage (or constrictor) is removed and a small dry aseptic compress is applied, being held in place by a gauze bandage. When possible the blood should be withdrawn by means of a good-sized cannula or aspirating syringe, as in collecting blood for a Wassermann.

DRY COLD AND HEAT

Cold and heat are widely used as therapeutic measures in infancy and childhood, either by dry or wet applications to the skin. The wet applications are considered under hydrotherapeutics.

The ice-bag serves as the means for applying dry cold. In using the ice-bag care should be exercised not to allow the sweating of the bag to wet the skin, because if prolonged this may cause a local necrosis. This may be averted by placing a piece of oiled silk between the skin and the ice-bag. The ice-bag serves to relieve pain and to reduce temperature. For the former purpose it may be applied over various parts of the head and body and is of special value where there is local inflammation. The size of the ice-bag will be determined by the area over which it is to be applied. When used to reduce temperature the ice-bag is usually applied to the head; a second bag may be placed over the abdomen if the head bag is not sufficient. In very young and delicate children it should be cautiously used. At times the little patients become rebellious to it.

The rubber coil filled with ice water is another manner in which dry cold may be applied, and is of special advantage in inflammation of the joints.

Hot-water Bag.—Dry heat may be applied by means of hot-water bag or bottle. In the employment of the hot-water bag or bottle caution should be exercised to prevent burning of the skin. Dry heat is indicated to relieve pain and to elevate and regulate body temperature. For the symptomatic treatment of pain, particularly when referred to the abdominal cavity, dry heat may be advantageously used, and in inflammation of the glands and joints dry heat exerts a soothing influence.

In conditions of subnormal temperature, as in shock and in weak, delicate, and premature infants, dry heat applied to the body surface and particularly to the feet is a valuable therapeutic agent. As hyperpyrexia may be produced by excessive external heat, care must be exercised in the employment of dry heat.

Poultices are another means for relieving pain. They may be made of various substances, the most common being the flax-seed poultice composed of flax-seed meal and water; this is applied hot. In certain local infections hot poultices serve to hasten definite suppuration. Poultices, however, are too frequently employed in local inflammation and may advantageously be replaced by compresses or wet dressings. When applied to the thorax in an enveloping manner, they hamper the respiratory efforts and thereby serve as a considerable handicap to the patient, particularly to the very young and feeble.

The **hot-air bath** is used to produce free perspiration. It is especially indicated in nephritis. The patient lies nude in bed upon a woolen blanket, and is covered with another blanket which is elevated about 1 ft. by means of a wire screen or wicker contrivance. Only the head of the patient is allowed outside of the tent thus formed by the bed clothing which must be fastened snugly about the neck. The dry heat is produced by means of electric lamps or electric pads, or by hot-water bottles or bags of hot sand, so protected that the child cannot be burned. The ordinary flat-iron also may be used as a source of dry heat. Free perspiration is promptly produced by the hot-air bath. The duration of this bath may be from 20 to 30 minutes. Care should be exercised not to produce hyperpyrexia.

HYDROTHERAPY

The external use of water, plain or medicated, with or without soaps and at different temperatures, offers the widest therapeutic field, exclusive of drugs, in diseases of infancy and childhood. In health, the daily bath serves to prevent many abnormal conditions by assisting and stimulating the functional activity of the skin. In disease, in the form of compresses, packs, or baths, water serves distinct local or general purposes, depending upon the temperature and mode of employment. The external application of water usually has a beneficial effect except under some conditions in which it is contra-indicated, more particularly in certain diseases of the newly born and in some cutaneous and surgical affections.

Bathing, except under the foregoing conditions and in premature and congenitally weak infants until these are increasing in weight, should be a daily routine, and a healthy baby should be given a daily tub bath, after the cord has fallen off. Baths serve to maintain physical comfort in hot weather, and a sick or delicate child should be bathed daily. Illness is not a contraindication unless the bath has an untoward effect. On the contrary the effect of a bath upon the skin and its functions is very helpful, and in the delicate child it has a stimulating and exhilarating effect.

The best water for baths is either rain or river or distilled water. Hard waters are objectionable because they require softening before use for cleansing purposes. This may be accomplished with bran or oatmeal enclosed in a bag which is stirred about in the water. Soda, ammonia and borax have the effect of drying the skin by the saponification of its fat, and should not be used for softening the water. Glycerin if applied to the skin after washing renders the skin moist but sensitive. Frequent hot baths favor the exfoliation and cornification of the skin. Soaps also have some effect upon the skin. Neutral soaps made from pure fats are best. Superfatted soaps have a soothing effect upon the skin. Soaps containing free alkali exercise an injurious effect by removing the natural fats from the skin, while those containing free fatty acids act as irritants. Medicated soaps may be used for disinfectant and therapeutic purposes, their effect upon the skin depending upon the drug which they contain.

Hydrotherapy may also be applied by the use of compresses, packs, and baths.

Compresses.—Circumscribed cooling and heating of the skin is accomplished by the use of compresses. They must be changed frequently, however, so that uniform and constant temperatures are maintained. Wet compresses should be covered with some impervious material in order to prevent the wetting of clothing or bed clothing.

Cold Compresses.—Linen or cheese cloth folded in several thicknesses is immersed in cold or ice water (50 or 60°F.) and wrung moderately dry. The effect of cold compresses may be prolonged by placing an ice-bag upon the wet compress. They are indicated in all acute local inflammations to relieve pain, swelling, heat and redness.

Warm compresses are applied by identical means, the cloths being immersed in water about 100°F., wrung out, and applied to the skin surface. A piece of flannel or oiled silk or rubber tissue over the compress will prevent rapid cooling. A hot-water bag upon the compress will retard cooling. The compress should not be replaced before it becomes dry. After the last compress has been removed, the skin should be dried and warmly wrapped. Warm compresses are indicated in neuralgia to promote absorption and hasten resolution, to relieve local spasmodic conditions, to hasten suppuration, and to relieve stasis by accelerating the blood current.

Packs.—In the application of packs the technique is virtually the same as for compresses. The naked patient is wrapped in a sheet, large bath towel or other soft absorbent material which is moderately moist or saturated with hot or cold water. He is then placed upon a blanket under which should be a rubber sheet to protect the mattress.

In *the cold pack* the entire body is enveloped with a sheet wrung from water at a temperature of 100°F. Ice is rubbed over this covering, first in front then over the back, then over the entire trunk. The temperature of the patient should be watched so that too great a reduction will be averted. The child is allowed to remain in the pack for a varying time and is then rubbed again with ice, after which it is wrapped in the underlying blanket without removing the wet pack. The pack is removed in from 1 to 24 hours, the patient is dried and wrapped in another blanket. During the pack the head should be sponged with cool water, and heat, if necessary, is applied to the feet. Shock and fright are avoided if the cold pack is given according to this method. The cold pack is sufficient to reduce a moderately elevated temperature, in which condition it is an effective and valuable therapeutic measure.

The Hot Pack.—For the hot pack the sheet is wrung from water at a temperature of 100 to 108°F. The patient is wrapped in the sheet over which a blanket is rolled. The hot applications are changed at intervals of about 20 to 30 minutes until free perspiration is produced, or even for a more prolonged period, if desired. Nephritis is the chief indication for the hot pack.

The *mustard pack* for the production of counterirritation, as already described (page 283), serves also as a hot pack.

The Vapor Pack.—The child, wrapped in a sheet wrung from water of 70 to 90°F. is then enveloped in blankets. By the additional use of woolen blankets the patient becomes warm after the initial cooling and an effect similar to that of a vapor bath is produced. The vapor pack causes free perspiration and is of value in extreme nervousness.

BATHS

Many kinds of baths may be given, but as they differ much in their application and in temperature range, each type will be considered separately. For determining the temperature of a given bath the ordinary bath thermometer may be used.

A **daily bath** should be given from the date of birth unless contraindicated, as for instance in congenitally weak or premature infants or in infants with an excessive postnatal loss of body weight. Until the cord falls, the child should be bathed from a basin of boiled water at about 100°F. using castile soap. After the cord falls and the navel is healed the full tub bath may be given for not exceeding three minutes.

The temperature of the room should be about 75° or even 80°F. The temperature of the bath should be 98° to 100°F. As the infant grows older the duration of the bath may be increased to five minutes and the temperature reduced to 90°F. by the end of the first year. Needless to say, the room should bec losed during the bath and drying and dressing the baby should be quickly done.

Cold Bath.—In this a child is put into water having a temperature of 100°F. This temperature is gradually reduced by the addition of ice or cold water to 75 or 80°F. Water should be applied to the child's head also, and its body rubbed vigorously. The bath may be continued for from 5 to 10 minutes, after which the child is quickly dried and rolled in a blanket. A cold bath may be employed as an antipyretic measure but is not to be preferred to the cold pack.

Cold Sponging.—After removing all clothing, except the diaper in the case of infants, the child is placed upon a blanket and its body is sponged with equal parts of alcohol and water, or vinegar and water, for from 10 to 20 minutes, with occasional rubbing of the skin. The child is then wrapped in a blanket without being clothed. It may be necessary to repeat this cold sponging frequently if the child's temperature continues high. Cold sponging, besides serving as an antipyretic, is of great value in relieving nervous irritability even when the temperature is very much elevated. As an antipyretic measure it may not be as effective as the cold pack but it is less depressing, and owing to its sedative effect it frequently serves as a satisfactory substitute for an anodyne.

The **cold sponge or shower bath** is usually given in the morning before the first meal, but if employed later in the day should be given not less than two hours after a meal. The room should be warm. The child stands in a tub filled to ankle depth with warm water. Water is squeezed from a large saturated sponge or is poured from a pitcher, or a shower spray is employed, the water, at 40 to 60°F., trickling over the body, preferably over the nape of the neck first, then the shoulders and chest, the skin being rubbed meanwhile. The bath should not continue over one-half minute. Following the bath brisk friction of the body should be continued until a thorough reaction is produced. For the younger children a dip or plunge in water at 50 to 70°F. may serve the same purpose. After these baths it should be noted whether the child reacts properly. If the child remains pale, pinched or blue, the bath should not be repeated or it may be given with water at a higher temperature.

Cold sponge or shower baths are useful in neurasthenia and as a general tonic. They may be employed for children of all ages and as early as the eighteenth month if cautiously given. They are of special value in delicate children. Full expansion of the lungs follows upon the deep inspirations or the strong cry which such a bath frequently induces, and it is therefore of decided benefit.

Evaporation Bath.—This bath is of value for the reduction of temperature. The body is enveloped in a double layer of gauze or some other porous textile which is then moistened periodically with water at about 95°F. Evaporation is produced by an air current set up either with an ordinary fan or an electric fan. During the bath the child's extremities should be kept warm by hot applications. The evaporation bath is more efficient than sponging as it produces no fright or shock and causes little disturbance to the child.

The Sitz Bath.—The patient is allowed to sit in water sufficiently deep to cover the lower part of the trunk and upper portion of the thighs, the upper part of the body being covered for protection from exposure. The temperature of the Sitz bath should be from 85 to 95°F. and the duration from 3 to 10 minutes. Sitz baths may be employed for relieving headache, are particularly valuable in certain conditions of the genito-urinary system, but should not be given just before bed time as they produce congestion of the genital organs.

A **tepid bath** is given as a full tub bath at a temperature of from 85 to 92°F. It is particularly useful for its soothing and mildly antipyretic effect, particularly in the eruptive fevers. Its duration should be about 10 minutes.

Warm Bath.—The warm bath, at 92 to 98°F., given as a full bath for about 10 minutes, serves to promote sleep, for which purpose it is often more effective than drugs, and is of much value in certain nervous conditions.

Hot Bath.—The patient is placed in a full tub with the water at 100°F., which is gradually raised to 103° or even to 106°F. A thermometer should be used to guard against excessive temperature of the water and to avoid the danger of burning the child. During the bath the body of the patient is well rubbed and cold is applied to the head. The hot bath is chiefly used to promote reaction in cases of shock or collapse.

The Hot Vapor Bath.—The same procedure and technique is employed for this bath as for the hot-air bath. Instead of using dry air, however, moist heat or vapor is produced by means of a croup kettle or an ordinary kettle so arranged as to allow the steam to enter under the bed clothing. There is always some risk in giving this bath as a fire may result from ignition of the bed clothing from the heating element.

SPECIAL AND MEDICATED BATHS

Mustard Bath.—Powdered mustard is employed in the proportion of an ounce to a gallon of water. The mustard should be first rubbed up with water until a smooth paste is formed after which water is added to make a solution free from lumps. This is added to the bath water which should be at a temperature of 100°F. The nude patient is placed in the bath. The temperature of the water may be gradually raised to 103 to 106°F. The bath should be continued for five minutes and not more than 10 minutes, but may be repeated in an hour if necessary. This bath offers the most efficient means of bringing the blood to the surface in cases of shock, collapse, heart failure, or sudden congestion of the lungs or brain.

Soothing Bath.—A soothing bath may be satisfactorily given by adding pine-needle oil in the proportion of three to five drops to a gallon of warm water. Infusions of camomile, sweet flag, or other aromatic herbs may also be used for this purpose.

The Brine Bath.—Brine baths are not given for cleansing purposes hence the patient should first be given a cleansing bath. The brine is prepared by adding ordinary table salt or preferably sea salt in the proportion of 2 oz. to a gallon of water. The temperature should be not more than 100°F. nor less than 90°F. Friction should be continued throughout the bath which should last for 5 or 10 minutes. The brine bath acts as a tonic and is indicated for delicate, undernourished and poorly developed children and those with a scrofulous tendency. It is a stimulating bath, but it is contraindicated in intertrigo, eczema and inflammatory conditions of the skin.

The Soda Bath.—Bicarbonate of soda in the proportion of one and one-half tablespoonfuls to the gallon of water constitutes the soda bath. The temperature should be that to which the patient is accustomed. It should be continued for from 2 to 5 minutes and there should be no frictioning of the skin. This bath is of some service in prickly heat and to allay the irritation of certain skin diseases, such as urticaria. The body should be dried with soft towels without rubbing.

The Bran Bath.—Ordinary wheat bran is placed in a bag of cheese cloth or muslin and stirred about in the bath, about one quart of bran being used to 5 gal. of water, until the water resembles a thin porridge. The temperature of the bath should be that to which the patient is accustomed though the best results are obtained at from 90 to 95°F. The bran bath is indicated when the skin is delicate and when the ordinary water bath apparently causes irritation of the skin, in excoriations about the buttocks, in prickly heat, and in eczema. There should be no frictioning and the skin should be dried without rubbing.

The Starch Bath.—Ordinary laundry starch is used in the proportion of half a cup to 5 gal. of water. The temperature, indications, mode of application and drying are the same as for the bran bath.

Carbonic Acid Baths.—These baths are prepared by using special chemicals (usually two sets of powders, one a carbonate and the other some acid as sulphuric, acetic, or hydrochloric acid). The tub in which the bath is given should be porcelain lined. Carbonic acid gas from compressed tanks may also be used, the gas being allowed to flow through specially made mats placed at the bottom of the bath tub. The advantage of the latter method is that the flow of gas may be regulated. The temperature of the carbonic acid bath should be from 90 to 95°F., and it should last about five minutes. Gentle friction is applied to the body after the bath and the patient should rest in bed for several hours. These baths should be given every other day over a prolonged period to be of any great value. Carbonic acid baths are advocated in cardiac disease.

Astringent baths are specially indicated in moist skin eruptions. They may be prepared by adding to the water tannin or better a decoction of oak bark made by boiling a cup of pulverized oak bark for 30 minutes, in a quart of water, straining and adding the decoction to the bath.

Sulphur baths for itching conditions of the skin may be prepared by dissolving from 1 to 3 oz. of sulphurated potash in the daily bath. The younger the children the weaker the solution should be made. After the bath the skin is usually anointed with some neutral salve.

Disinfectant baths are usually prepared with mercury bichlorid 1:20,000. Potassium permanganate, though less effective for disinfection, also has an astringent effect, and may be employed in a strength of 1:5000 to 1:3000. For a potassium permanganate bath the water must be distilled, which is a disadvantage in ordinary practice.

Medicated soaps are used in connection with the ordinary baths for the special effect of the ingredient contained in the soap.

Natural Baths.—These baths are of two general classes water and peat (or mud) baths. The latter are not indicated in the treatment of diseases of childhood. The former comprise certain natural spring baths found in various parts of the country, including cold, warm and hot baths. Cold spring baths are not indicated for children. The warm baths are particularly valuable for weakly children and those of neurasthenic tendencies, while the hot spring baths are especially indicated in rheumatism. The presence of carbonic acid and mineral substances in the waters further enhance their therapeutic value.

Hypodermic medication in infants and children does not differ in application from its use in adults. As a rule it is not employed in early life except for serious conditions. The danger from idiosyncrasies must not be overlooked. In older children, because of the fright and psychic shock incident to the pain, this method should be adopted only when necessary. Hypodermic medication is practiced more particularly for the administration of sera, vaccines and drugs, the latter when prompt action is desired, as for example: Morphin in convulsions, and morphin and atropin in cholera infantum. Other drugs which may be administered with advantage by this method are camphor, caffein, digitalis, epinephrin and strychnin.

Hypodermoclysis, which consists in the introduction of fluid beneath the skin (Fig. 111), is one of the most valuable therapeutic measures, especially in infancy, when the prompt and certain introduction of fluid is necessary. The apparatus should be sterilized and the procedure carried out under strict aseptic precautions. The apparatus consists of a container to which is attached a rubber tube to carry the fluid to the hypodermic needle at the other end of the tube. By the use of a glass Y-shaped connecting-tube and several short sections of rubber tubing, two hypodermic needles may be connected with the apparatus. Usually normal salt solution is employed; this should be injected at body temperature. The quantity administered may be from 1 to 4 oz. for young infants, and larger quantities, up to 8 oz., for older children. The injection may be repeated two, three, or more times during the 24 hours depending upon the rapidity of absorption, bearing in mind, however, the daily fluid requirement of the individual. The fluid should not be injected too fast as the distention incident to the entrance of the solution into the tissues may cause much discomfort and pain. From one-half to two hours may be allowed for completing the procedure. The best site for introducing the solution is over areas containing considerable loose areolar tissue: The abdomen, the lateral thoracic regions, and the back between the scapulæ are most frequently selected. A slight rise of temperature may follow hypodermoclysis.

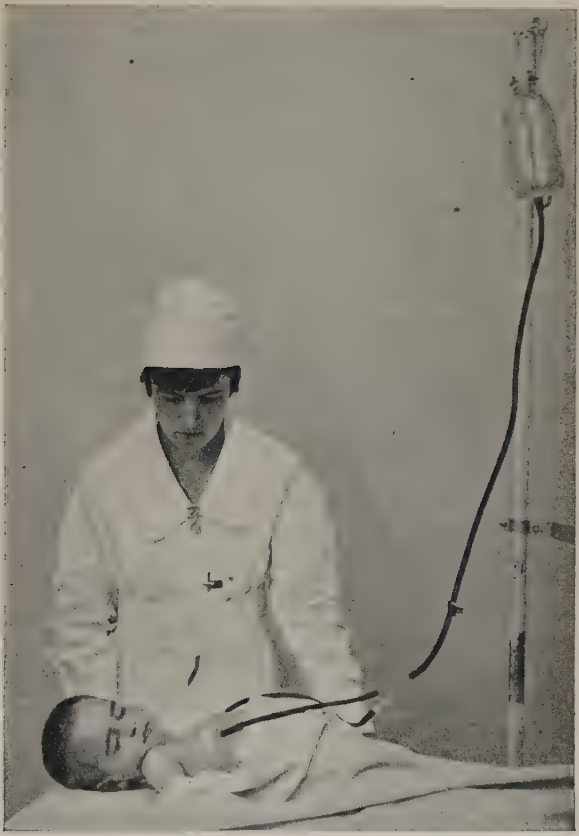

Fig. 111.—Hypodermoclysis.

Hypodermoclysis is indicated in great loss of body fluid (to compensate for this loss), in a deficiency of fluids (to give added fluid) and when the general circulation requires stimulation. As this method may be employed for several days, it has a decided advantage over the introduction of fluids through the rectum.

Intravenous Injections.—This valuable therapeutic procedure has until recently been neglected owing to the relatively small size of the veins in the infant and small child and the consequent difficulty of entering the vessels. During the first 12 or 18 months of life the veins are very small and not until after this age are they of sufficient caliber to permit ready perforation. Various superficial veins of the body have been used for intravenous injections, including those at the bend of the elbow, and in the neck and scalp. More recently, in children with an open anterior fontanel, the longitudinal sinus has been utilized not only for securing blood for diagnostic purposes when relatively large quantities are necessary but also as a favorable site for intravenous injections.

When it becomes necessary to expose a vein for intravenous infusion the ordinary method is employed, a small cannula, however, facilitates the procedure. When the veins are sufficiently large so as not to require operative exposure, or when the longitudinal sinus is utilized, the skin should be thoroughly disinfected. The apparatus, which should be sterilized, consists of a funnel or glass container, rubber tube and a large needle. It is superfluous to observe that an intravenous infusion should be carried out aseptically as the likelihood of introducing infectious material obviously presages disastrous results. When the quantity to be injected is small and can be contained in a small syringe, no apparatus is required other than the syringe and needle. For intravenous infusion, normal sodium chlorid or sodium bicarbonate solution, or 5 per cent. dextrose solution, or drugs may be employed. Of the latter salvarsan and neosalvarsan are most frequently given by this method. The possibility of damage from the introduction of air should be kept in mind. When salvarsan or neosalvarsan is administered it is advisable to introduce a normal saline solution first and to add the drug to it.

When the longitudinal sinus is selected as the site for infusion a syringe may be employed, as advocated by some authors, because of the advantage of determining by aspiration when the needle is in the vein. The barrel of the syringe is connected to a short piece of rubber tubing which in turn connects with a glass tube (to serve as a window), the other end of which slips into a second piece of tubing. A large needle, for introduction into the vein, is attached to the distal end of the second section of tubing. It is more advisable, however, when utilizing the longitudinal sinus, to make the injection by the gravity method rather than with a syringe. If a syringe is employed the fluid for injection is drawn into it and all air is expelled. An assistant should hold the head of the infant. The area over the fontanel having been sterilized, the needle is introduced in the median line at the posterior angle of the fontanel. As soon as the longitudinal sinus is entered a lessened resistance will be noted. While the operator holds the needle steady, another assistant slightly withdraws the piston of the syringe, when entrance of blood into the glass tube will indicate if the needle has entered the vein. The piston is then gently forced into the syringe and the contents injected very slowly. The quantity should not exceed one-sixteenth of the infant's body weight and the injection should require from 10 to 20 minutes. If the veins of the scalp become distended it indicates that the injection is proceeding too rapidly.

Transfusion.—This therapeutic measure is relatively rarely employed in the treatment of diseases of children, but under certain indications is of great value. The indications for transfusion are: (1) The hemorrhages of the newly born; (2) acute hemorrhages; and (3) some forms of severe secondary anemia.

Various methods and apparatuses have been advocated for carrying out this procedure, but most of them are distinctly surgical and require a skilled surgeon. However, where feasible, the syringe method may be employed satisfactorily by a physician with only limited surgical experience. In those conditions in which transfusion is of greatest benefit, that is, in the hemorrhagic disease of the newly born and after spontaneous hemorrhage, the intravenous introduction of blood through the skin without exposure of the vein is preferable because any cutting will only leave a larger surface exposed to the danger of hemorrhage.

In transfusion the blood must be passed rapidly from the donor to the recipient before coagulation takes place. For the syringe method, several syringes are used and an assistant is necessary. The skin surfaces must be rendered aseptic; the blood is withdrawn from the donor into a paraffin-lined glass syringe and immediately injected into the vein of the child. Any available vein may be utilized; in infants, before the anterior fontanel is closed, the longitudinal sinus offers the most favorable route of injection. The donor should preferably be a member of the family or, if not, the blood of the donor should be tested for its hemolytic action with the blood of the recipient. For transfusion in the hemorrhagic disease of the newly born only the blood of a parent should be used without previous hemolytic tests. The amount of blood to be injected at a time varies from 2 to 6 oz.; the transfusion may be repeated as indicated, every few hours, if the hemorrhage is not controlled.

ELECTROTHERAPY

In pediatrics electricity may be employed for diagnosis, prognosis and treatment. For diagnostic and prognostic purposes, electricity is of decided value but for the treatment of disease it is very much overrated. For utilizing electricity in medicine, a knowledge of its fundamental laws, of the use of instruments, and of the proper interpretation of results is a prerequisite.

The faradic induced or interrupted current, and the galvanic continuous or constant current are usually employed. The faradic current is used for its mechanical property of stimulating muscular contractions or as a counterirritant, inasmuch as it possesses little or no cataphoric or electrolytic action. The galvanic current, on the other hand, is selected whenever cataphoric, electrolytic and electrotonic effects are required, with or without marked sensory and muscular stimulation.

Electricity is applied by the polar method, using two electrodes, a smaller one—the exciting or localizing electrode—measuring three square cm., and a larger one—the indifferent or dispersing electrode. The exciting electrode is placed over the nerve to be tested and the indifferent electrode at as great a distance as possible from the nerve. The greater the surface of the indifferent electrode the more reduced will be its local action, even to complete elimination of action. The purpose of the indifferent electrode is, therefore, simply to complete the circuit. By the use of a pole changer the current may be reversed, the small electrode being made to constitute either the negative (cathode) or positive (anode) pole. An interruptor in the handle of the instrument serves to make and break the current. When increased excitation or stimulation is desired the cathode is used as the active electrode and is applied over the locality to be treated, but when a sedative effect is desired the anode is used as the active electrode. The polar method applies only to the galvanic current. The normal sequence of reactions with this current is as follows: Beginning with the weakest current, there follow in order (1) cathode closing contraction; (2) anode closing contraction; (3) anode opening contraction; (4) cathode opening contraction. The contractions are transient even during the flow of a current of medium strength; with a very strong current, however, the cathode closing contraction may be continuous—a tetanus.

It should be remembered that the larger and more massive the muscle the slower is its contraction. In the use of electricity for therapeutic, as well as for diagnostic and prognostic purposes, the current density and certain other factors should always be known. The current strength (1) varies in the production of the first visible contraction in different nerves and muscles; (2) it varies for different nerves and muscles; (3) it varies for different individuals, and (4) it may vary in the same individual at different times. The current strength, expressed in milliamperes, is determined by measuring the least amount of current necessary to produce the first visible contraction. The current density is equal to the strength of the current divided by the area of its conductor. The weaker the current necessary to produce the contraction the greater the excitability of the contracting muscle, and vice versa.

In establishing measurements comparative observations should be made of the opposite sides of the body. The polar law applies to the galvanic current but not to the faradic current. In medical application, the poles are used indiscriminately with the faradic current. The contractions produced by the faradic current are tetanic, in other words, the muscles contract at the moment the current is allowed to pass through the tissue and remain in contraction until the flow of the current is interrupted.

While electricity is used mostly for diagnostic and prognostic purposes its therapeutic value in pediatrics lies principally in its stimulating effect upon the muscles and nerves. However, it has also been advocated for a certain psychic influence, as, for example: hysteria.

In applying electricity in pediatrics extreme caution and circumspection should be observed. The confidence of the little patient should first be secured and all fear eliminated. Undue haste and lack of consideration of the patient will result in the development of antagonism and resistance that will greatly discount any beneficial influence which might otherwise result. The coöperation of the patient is essential.

The frequency of application and the duration of treatment should be influenced by the character of the disease, with special reference to whether it is acute or chronic. There is a decided tendency to give too prolonged treatments in electrotherapy. The duration of the application should be governed and recorded by the clock. Local applications may require from two to five minutes, and general applications from 10 to 15 minutes. Electricity may be applied directly or indirectly. In the former the electrodes are placed near the area to be treated; in the latter method they are placed at some distance from the involved area.

For polar effects the direct method should be employed. If this is impossible, a knowledge of the relation of the viscera to definite cutaneous areas is essential in order that the proper nerves may be stimulated. Other important factors in the application of electricity are the size of the electrode and the dosage employed. The smaller the electrode the more local the effect and the less deep the penetration; on the other hand, the larger the electrode the deeper the effect of the electricity. The galvanic current possesses decided advantages over all other electric currents, mainly because the strength and density of its current can be accurately regulated.

The dosage strength of the faradic current can be expressed only in terms of strong, medium, and weak, with reference to a particular or standard apparatus. The dosage of static and high frequency currents is difficult to predetermine; these currents are of little value, particularly in pediatrics.

RADIOTHERAPY AND ROENTGENOTHERAPY

The term "radiotherapy" was formerly applied to the therapeutic use of any radio-active substance, including both radium and the Roentgen-rays. However, latterly the term has been reserved to signify the therapeutic application of radium, while Roentgenotherapy applies to the therapeutic uses of the Roentgen-rays.

In the use of either method experience is essential for success. Numerous technical details are involved and these require the expert services of one conversant with this branch of therapeutics. Radiotherapy and Roentgenotherapy are apparently simple in application, but it must be emphasized that while each is a valuable agent when properly and intelligently used, both are capable of infinite harm to the patient and to the operator if inexpertly employed.

The therapeutic field of these agents is somewhat similar and in many conditions the desired results can be obtained with either. In general, radium is more suitable for limited superficial conditions, whereas the Roentgen-rays are more suited to extensive or deep lying conditions. There are, of course, many exceptions; thus radium is much more satisfactory in the treatment of a malignant tumor even though deep, provided the element can be embedded directly in the tumor mass.

Radium is employed either as a salt, as the bromid or sulphate, or in the form of emanation derived from the salts in solution through a complicated physical process. The mode of application varies with the condition under treatment. For a superficial process, such as a nevus, the radium is applied through an appropriate filter directly over the growth. For treating a deep nodule of malignant origin the radium may be buried in the tumor mass, in the form of needles or small tubes. The question of filtration (by metallic filters, rubber, gauze, etc.) is determined by special factors in the individual case. The same applies to dosage, regarding which no hard and fast rules may be laid down. There are no contra-indications to radium treatment unless the patient has an idiosyncrasy, which can be determined only by the reaction after the first treatment. Radium must often be administered over a long period of time, for even with the utmost care it is rarely possible to administer safely a curative dose at one sitting. With intelligent and careful use, radium possesses no real dangers.

The main indication for the use of radium in pediatrics is for the treatment of various forms of nevi, in which success is practically certain. Similar success is also obtained in the treatment of superficial papillomas and of keloids. In the malignant conditions of childhood, radium should usually be tried.

For Roentgenotherapy an elaborate apparatus is needed, in contrast to the few simple unimpressive tubes for radium application. Practically, the only form of machine now used for Roentgenotherapy is the interrupterless transformer type of ample capacity to give the powerful current required for modern therapy, with either a gas tube or the newer so-called Coolidge tube (virtually a gas-free tube).

The application of the Roentgen-ray entails considerable technical detail. In general, the same problem is involved as with radium, that is, the application of the desired dosage to the affected part without injury to the healthy tissues. This is accomplished by proper filtration, adequate protection of the healthy tissues and a careful control of the dose. Treatments may extend over a long period of time. The dangers to the patient are practically nil if the applications are carefully and intelligently made.

The field of Roentgenotherapy in childhood is rather wider than that of radiotherapy, and includes in addition to the conditions suitable for radium treatment the chronic skin diseases which are often resistant to other treatment. One of the most important of this group is ringworm which is treated with uniform success. Roentgenotherapy is also beneficial in glandular conditions, especially enlarged lymphatic glands, and is worthy of trial in Hodgkin's disease. At least a temporary improvement may be obtained in pernicious anemia and the leukemias. The treatment is also advocated in conditions of enlarged thymus, but it should be remembered in this connection, that the application of the Roentgen-rays may result in a transient increase of the symptoms due to temporary stimulation.

VACCINE AND SERUM THERAPY

Much confusion exists among the laity regarding the terms "serum" and "vaccine." In a great measure the responsibility for this is due to the loose manner in which these terms are usually employed by the physician. Objections are occasionally raised when it is desired to use "vaccines" for fear that some possible harm may ensue, the confusion of the patient being due to the possibility of anaphylactic shock or serum sickness, which may occasionally accompany the use of a serum. On the other hand there is frequently a reluctance on the part of patients to submit to the use of a serum of proved value because of doubt as to the beneficial effects, owing to the uncertain results that more frequently accompany the use of the vaccines. The danger incident to their administration is negligible if proper asepsis is used.

Serum Therapy

Serum therapy embraces the employment of a serum as a therapeutic or prophylactic agent. The serum may be derived from a human being or from some domestic animal. It may be useful merely for its chemical or physiologic properties alone, as for example: In the treatment of certain hemorrhagic conditions, or it may be used in the form of a specific serum containing the immunizing bodies.

The preparation of artificial immune sera is based on the principle, that the inoculation of bacteria, either living or dead, or their products, causes the elaboration in the inoculated animal of certain substances which are antagonistic to the injected bacteria; these substances are known as immune bodies. The immune bodies comprise several different "antisubstances," such as the antitoxins, the agglutinins, the bacteriolysins, and opsonins, which by concerted action tend to suppress bacterial growth within the body, or to neutralize the harmful effects of bacterial activity. The antitoxins neutralize the toxins of the bacteria; the bacteriocidins and bacteriolysins kill and dissolve the bacteria; the agglutinins tend to inhibit the motion or progress of invasion of bacteria, and the opsonins render the bacteria peculiarly attractive to certain cells of the body fluids for ingestion and destruction.

Those bacteria which produce a soluble toxin as a by-product of their growth and multiplication yield sera of highest potency for prophylaxis and therapeusis. While many sera are available, only a few are of proved value, among these being the antidiphtheritic, the antitetanic and the antimeningococcic sera. Other sera which have been employed include those against the streptococci, the pneumococci, the bacillus of influenza, the tubercle bacillus, and the dysentery bacillus.

From a clinical point of view, sera may be divided into three classes: (1) Those which are of proved therapeutic value, such as the antidiphtheritic and antimeningococcic sera; (2) those which are of proved prophylactic value, such as the antitetanic and antidiphtheritic sera; and (3) those which are of unproved clinical value.

The uses and administration of the various sera will be considered in connection with the discussion of the respective diseases. It should be said in emphasis, however, that there is a great tendency to employ sera in too small doses. Large doses are of decided value in shortening the course of a disease and in considerably minimizing the complications and sequels. It is hardly possible to give too large a dose of serum.

The principal objection advanced against the use of sera for therapeutic and prophylactic purposes is the probability of sensitizing the individual to the serum of the particular animal (usually the horse) used in the artificial production of immune sera. To prevent acute anaphylactic shock in cases in which the administration of serum is to be continued over a long time it is advisable to give some serum daily so as to prolong the period of desensitization, as, for example: Meningitis.

That the removal of the immune bodies with the globulins from the major portion of the serum renders the so-called "globulin fraction" slightly less liable to cause sensitization is claimed by some authorities. This serum is also asserted to be less toxic to a sensitized person. As a matter of fact, a small dose of serum alone will produce either sensitization or a reaction in a sensitized individual, and the globulin fractions always contain enough serum to produce these results.

Before the administration of a serum it should first be determined whether the individual has already received a similar animal serum or is sensitized to the animal serum to be administered. If this is found to be so, and the serum is nevertheless essential, the patient should be desensitized as follows: A fraction of a drop of the serum is injected intradermally, and if in a half hour there is no local anaphylactic reaction, a larger quantity is given subcutaneously. If this is followed by no evidences of disturbance either in the pulse or respiration, the full dose may be administered in another hour.

The principal advantage of serotherapy is that it introduces into the organism certain immunity substances to elaborate which the body cells would require many days. These substances tend to render milder or to shorten the course of the disease. Sera confer a passive immunity. Serum may be injected intramuscularly or intravenously, and in meningitis into the spinal canal or lateral ventricles.

VACCINE THERAPY

Vaccine therapy consists in the introduction of bacteria usually dead cultures) into the organism, for the purpose of prophylaxis or therapeusis. By this inoculation the body cells themselves are stimulated to the formation of the immune bodies. The vaccines are, therefore, of greater value in the prevention than in the cure of disease. Certain vaccines do, however, exert beneficial clinical effects in infectious processes, more particularly in those which are distinctly localized or are characterized by chronicity, and where comparatively long periods of time are not a factor.

Vaccines may be considered to be of three varieties: (1) Viruses, as the living unknown organism of cow-pox which is inoculated into an abrasion of the skin for prophylaxis against small-pox. (2) Bacterial extracts, such as tuberculin, which is derived from the tubercle bacillus. (3) Suspensions, consisting of killed or attenuated bacteria in some diluent (water or salt solution), as the typhoid vaccines.

The vaccins of Groups 1 and 2, being more specific, will be considered under the respective diseases against which they are employed. The last group constitutes by far the greater variety of vaccines and will be given general consideration here.

It has been definitely shown that killed bacteria do not lose their power of stimulating the production of immune bodies when inoculated into an individual, but being dead, they cannot multiply nor produce disastrous consequences.

The suspension of killed bacteria may be either an autogenous or a stock vaccine. For prophylactic purposes, stock vaccines must of necessity be employed; but for therapeutic uses an autogenous vaccine is undoubtedly preferable whenever it can possibly be prepared. While bacteria may be readily isolated, detected and classified from a morphologic standpoint, these organisms may be further divided into various strains which differ only in the specificity of the immune bodies produced on inoculation. Thus an autogenous vaccine, besides producing the ordinary immunity to such organisms, has also a more specific influence. It should be remembered that the fresher the vaccine the more effective the results that will follow its use. Many failures in vaccine therapy may be ascribed to ignoring this factor. The strength of the vaccine depends upon the number of killed bacteria in a given unit, and is usually measured by the number of bacteria per c.c. The dose varies with the type of vaccine and with the purpose for which it is to be employed, whether for prophylaxis or therapeusis. The size of the patient and the special reaction to the vaccine must also be taken into account.

For prophylactic purposes, vaccines have been most widely employed in typhoid and the paratyphoid fevers. It should be borne in mind that the immunity conferred by the vaccine is not as lasting as the immunity that follows an attack of the disease itself. Except probably against the aforesaid diseases, prophylactic vaccination is indicated only during epidemics and after exposure to certain diseases. Vaccines have been employed as a therapeutic measure in almost every kind and condition of bacterial infection. The results have been rather generally disappointing, particularly in conditions of general systemic infection. On the other hand, when the infection is localized, favorable results are frequently obtained. Vaccines should not be given at too frequent intervals for fear of overcoming the ability of the body cells to produce the immune substances, thereby lessening the resistance of the individual. The rationale of the use of a vaccine for therapeutic purposes is that it stimulates the body forces to increased effort, and it should be remembered that care is to be taken not to over-power these forces by additional toxic substances.

The immunity produced by the use of vaccines is both active and passive; that is, a present store of antibodies is produced, and the fixed tissue cells that have once been "taught" to produce a given specific antibody reacquire that function in the presence of a new infection much more quickly than "uneducated" or non-sensitized cells. The mildness of an attack of typhoid fever following a prophylactic inoculation some time previously is thus accounted for. The indications for the various vaccines will be considered in the discussion of each individual disease.

PHARMACOTHERAPY

In the treatment of the diseases of infancy and childhood pharmacotherapy occupies an important place. Undoubtedly drugs are too frequently prescribed when other measures may serve the same purpose as well, if not better. Particularly is this true when the members of the family or friends kindly advise medication. This is frequently done in order to impress parents, and the consideration of the patient's welfare is apparently of secondary importance. Only too often the physician is called only after the patient has already been over medicated. The promiscuous use of drugs may at times result disastrously.

Drugs unquestionably serve a distinct purpose and a thorough knowledge of a few drugs is vastly to be preferred to a meager knowledge of a great number of them. A drug should never be prescribed unless its use is clearly indicated, and if its toxic effect follows its administration, the drug should be discontinued for a time, when it may be readministered in smaller doses. Should there be no effect from a given remedy, some other should be employed.

Polypharmacy is bad practice. Drugs should be given singly or in simple combinations. The time of and intervals between administration, with special relation to meals, should be taken into account when prescribing. Drugs should be used as little as possible for infants during the first six months of life. In administering them to children several points should be remembered: (1) The proper drug should be selected. (2) Its dose should be accurately determined. (3) It should be palatable if it will serve the same purpose as an unpalatable one. (4) It should be prescribed in as palatable a form as possible. (5) It should not be given with a food if this may subsequently cause a distaste for the food. (6) Its idiosyncrasies and toxic effect should be borne in mind. (7) Many highly seasoned syrups are administered with greater difficulty than some bitter drugs diluted with water. (8) It should be ascertained which drugs are well borne and which are not. (9) Drugs should be given frequently in small doses rather than occasionally in large doses. (10) A medicine should be given in such a form as to be easily swallowed. (11) Liquids are preferable to powders or pills. (12) Simple solutions, concentrated tinctures, fluidextracts, alkaloids and their salts, should be given by preference in the order named. (13) A medicine glass is more accurate than the domestic spoons (which vary greatly in size) for measuring doses of liquid medicaments. (14) Drugs in tasteless combinations, or in form of exact dosage are of especial value for children. Tablets may be dissolved before administration.

The drugs which are well borne by children include alcohol, belladonna, the bromids, chloral, digitalis, the iodids, all mercurials (including calomel) and quinin. The drugs which are not well borne may be considered under two categories: (1) the toxic drugs, as antimony, apomorphin, cocain and heroin; and (2) the irritative drugs, that is, those which have a tendency to cause gastric disturbances as, for example: the acids, astringent preparations of iron, etc.

In the consideration of the various drugs here, it is assumed that the physician is familiar with their source, general description and action, as well as their elimination and toxic effects, and the contraindications to their use. While the drugs under discussion may have other pharmacologic action they will be considered more particularly for their beneficial and most important effect in the treatment of children.

Drugs Deserving Special Mention.—Certain drugs require special consideration in their application to pediatrics. Their dosage appears in the table of drugs on page 320.

Adrenalin, as adrenalin chlorid in solution of 1:1000, is useful in conditions of circulatory collapse due to vasomoter disturbance, and in asthma and urticaria. It may be given by mouth or hypodermically.

Alcohol, which is well borne by children, is useful in many conditions, though it is misused and abused at times. It is of particular value: (1) in conditions in which there may be cardiac failure, as in certain eruptive fevers, bronchopneumonia, and diphtheria; (2) in conditions of shock or collapse; (3) in conditions of malnutrition; (4) in many chronic conditions; (5) in conditions of exhaustion or slight debility; (6) in certain forms of diarrhea, and (7) late in acute febrile conditions. It is contra-indicated in acute febrile conditions of short duration when the heart action is strong and the temperature high.

It is administered usually in the form of wine and spirits, the most common forms being whisky, brandy, port wine, and champagne. Whisky and brandy have an alcoholic content of from 40 to 45 per cent. The dosage of the various wines should be computed upon the alcoholic strength of whisky. The alcoholic preparation should be given either well diluted with water (one part alcohol to six parts water) and slightly sweetened, or added to the milk food. It is best given at a feeding time.

Alkalies and alkaline salts are well borne and serve a valuable purpose in neutralizing acidity. Lime water and bicarbonate of soda are particularly useful to overcome gastric acidity, and are used for the modification of cow's milk for infant feeding. The citrates and acetates of potassium and sodium are used in febrile conditions as are also potassium citrate and solution of ammonium acetate. The alkaline salts are frequently used in cough mixtures and diuretic mixtures, in the latter to render the urine alkaline. In conditions of acidosis, sodium bicarbonate is given as a means of increasing the alkalinity of the blood.

Antipyretic Drugs.—Acetanilid, antipyrin, and phenacetin, although frequently classed as drugs that are not well borne by children, have, in the writer's experience, been of especial value and decidedly well tolerated if properly administered, particularly when combined with small doses of caffein. Of the three, phenacetin, though not so readily soluble, has a less objectionable taste and is more easily taken. These drugs should be cautiously given, acetanilid probably requiring closest attention. They should not be given in large doses for the reduction of temperature, but are particularly useful when external antipyretic measures are not well borne. They should not be given to produce a decided and sudden drop in temperature. When this does occur in conjunction with much sweating the drug should be discontinued.

It should be remembered that a sharp elevation of temperature in a child, without untoward symptoms, need not cause any alarm in itself, and that children often tolerate prolonged fevers well. In hyperpyrexia and when there is much discomfort, restlessness, headache, pain or threatening symptoms, as delirium or impending convulsions, the coal-tar products are of particular advantage. They are also indicated in controlling nervous symptoms in the absence of fever and in certain spasmodic affections, as asthma, chorea, pertussis, etc. In these conditions larger doses than those ordinarily given are often exceptionally well tolerated. Smaller doses should always be given at first, and may be increased as the patient shows a tolerance for the drug.

Acetylsalicylic acid, quinin and sweet spirits of niter (spiritus ætheris nitrosi) are also used as antipyretic drugs. The effect of acetylsalicylic acid is very much like the three drugs already mentioned. Its use is somewhat abused. It is probably better than the foregoing drugs in infectious conditions, particularly in those of the upper respiratory tract, and in rheumatism. Quinin is indicated in malaria. Sweet spirits of niter is beneficial in mild fevers, especially when elimination is at fault. Its advantage in the reduction of fever is due to its diaphoretic and diuretic effects.

Aromatic oils are useful as vehicles for drugs and are usually administered in the form of waters as, for example: Cinnamon, peppermint and spearmint oil. The latter is more acceptable to infants than the peppermint water, which is too strong.

Arsenic is well borne by children. The principal form in which it is employed is as Fowler's solution (liquor potassii arsenitis). It is used as a gastric sedative in some forms of anemia, in syphilis and chorea, etc. Care should be exercised in the use of this drug, inasmuch as its over-action may cause peripheral neuritis. The urine should always be watched for evidences of irritation when this drug is prescribed.

Belladonna and Atropin.—One drop of the tincture of belladonna is equivalent to 1:3000 of atropin. The drug is particularly well borne in early life, when the dose may be comparatively larger than in later life. Idiosyncrasies to the drug must not be overlooked, hence smaller doses should be prescribed at first. The drug is used particularly in the treatment of enuresis; in conditions in which there is excessive secretion from the respiratory mucous membrane, and in combination with morphin to overcome certain effects of the latter.

Bismuth, an astringent and antacid, is used chiefly in treatment of certain forms of diarrhea. When prescribed it should be given in sufficient quantity to produce the desired result. The subnitrate or one of the various suspensions such as the milk of bismuth, lac-bismo, etc., may be given.

Bromids are especially well borne and are the most desirable sedatives in early life. Very much larger doses may be given to an infant than are prescribed for the adult. The sodium salt is usually employed. Bromids are indicated in the functional nervous disturbances, as insomnia, etc., as well as in the more serious nervous disorders, as for example: Convulsions. The drug should always be given well diluted when administered by mouth. It may also be given by rectum.

Caffein is a valuable circulatory stimulant as it acts both as a vasomotor and cardiac stimulant. It is also useful as a diuretic. It may be given in the form of caffein sodium benzoate or caffein sodium salicylate, and may be administered hypodermatically or by mouth. Caffein citrate is to be given by mouth.

Camphor (as the oil) is given either subcutaneously or intravenously for its rapid stimulating effect in conditions of sudden circulatory collapse.

Chloral which is well borne by children, is undoubtedly the most valuable drug for the treatment of convulsions. It is best given well diluted, preferably by the rectum as it is occasionally irritating to the stomach. It has marked narcotic effects, hence, its administration should be closely watched.

Cod-liver oil is not only one of the best available tonics but also a food. It may be given as the pure oil or in emulsion, or in various combinations with malt and wine. If there are gastric or intestinal disturbances it is probably not advisable to prescribe it. As with all oils in hot weather, cod-liver oil may become rancid, it should, therefore, be kept in a cool place. Usually the clearer the oil the better it is.

Creosote is used particularly in pulmonary affections, because it is eliminated for the most part by the bronchial mucous membrane. In prescribing it, beechwood creosote should be specified, either in emulsion or in combination with cod-liver oil.

Digitalis is a useful drug, more particularly in the conditions of cardiac insufficiency due to diseased conditions of the endocardium, myocardium, or pericardium. It is more valuable in the chronic than in the acute diseases of the heart. The action of digitalis sets in slowly but because of the cumulative effect the medication should be closely controlled. Various preparations of digitalis are perfectly satisfactory for hypodermic as well as oral administration. Those more commonly used are digilin, digipuratum, digitalone, digitalin, and digifolin.

Hexamethylenamin, known under the proprietary name of urotropin, is used particularly in the treatment of pyelitis and cystitis, and in typhoid fever to prevent urinary infections due to the typhoid bacillus. It is useless if given when the urine is alkaline as it is eliminated unchanged, but with an acid urine it is eliminated in the form of ammonia and formaldehyd. The effectiveness of the drug depends on the elaboration of formaldehyd. If it is desired to administer hexamethylenamin when the urine is alkaline, acid sodium phosphate should first be given to render the urine acid. These drugs should not be prescribed together as they are imcompatible.

Iodids.—These are well borne by children. The preparations most frequently used are those of the sodium and potassium salts, the syrup of hydriodic acid and the syrup of the iodid of iron. They are used as expectorants and alteratives especially in tertiary syphilis. The iodids are also useful in glandular enlargements and in asthma. Any over-action is shown by rhinorrhea, headache, papular or erythematous eruptions and gastric disturbance.

Iron is one of the most important elements in the composition of the body. It is taken in the form of organic compounds in the various vegetables, eggs, meat juice, etc. At times it becomes necessary to introduce iron into the system through the agency of the various inorganic salts. Iron is particularly useful in the anemias. It may be given subcutaneously in the form of the citrate (marketed in ampules containing one dose ready for use), a platinum needle being necessary in its administration. It may also be given by mouth in the various preparations, such as the lactate, citrate, pyrophosphate, saccharated carbonate, the dried ferrous sulphate, tincture of ferric chlorid, syrup of iodid of iron, etc. In older children it may be given in the form of Blaud's pills or Vallet's mass. In giving iron by mouth the possibility of damage to the teeth and disturbance of digestion should not be forgotten.

Ipecacuanha is a very useful drug in pediatrics, being employed as an expectorant and emetic. As an expectorant it is particularly good in croup, and in larger doses, as an emetic, it is one of the most serviceable, but is very slightly depressing in its action. The wine and the syrup are the commonly used preparations. Ipecac is also indicated in amebic infections, for which it may be given either perorally in a salol-coated pill or in the form of its alkaloid, emetin, by injection.

Malt preparations, either alone or in combination with cod-liver oil, are at times of value as tonics. They are frequently well borne when cod-liver oil is not tolerated.

Mercury is a very valuable drug in infancy and childhood and all of its preparations are well borne. It is principally used: (1) In congenital syphilis; (2) as a purgative; and (3) in the treatment of certain skin diseases. In congenital syphilis it is employed by inunction in the form of blue ointment (mercurial ointment) with equal parts of vaseline in winter or lanolin in summer, or internally as calomel, grey powder, or the bichlorid. Mercury is given as a purgative in the form of the mild chlorid (calomel). In skin affections mercury is of great value, particularly in impetigo.

Opiates are sometimes absolutely necessary, but when prescribed caution should be exercised lest the indiscriminate use have ill effects. Especially in the first few weeks of life great caution should be employed. Opiates are less often required in infancy and childhood than in later life. As they are not well borne by children very small doses should be given, and the weaker preparations should be employed. These should be repeated only if necessary. Opium is particularly powerful in impending coma or in debilitated conditions.

Codein, Dover's powder, morphin and paregoric are most commonly used. These are indicated to overcome spasmodic conditions, to diminish peristalsis, to allay pain, and to produce hypnosis. Codein has the same effect as morphin but is milder and is also less constipating. Dover's powder is especially useful for young children to relieve cough, to check certain forms of diarrhea, and to control pain. Morphin is the best preparation for controlling severe pain. It may be given either by hypodermic injection or by mouth. Paregoric has an effect similar to Dover's powder and is used for the same indications.

The other preparations of opium in use are the deodorized tincture and heroin. Opium is a valuable drug when properly administered and if judiciously employed it may be prescribed without fear.

Phenacetin is considered in connection with the antipyretic drugs.

Potassium chlorate is used in the various forms of stomatitis, both as a mouth wash and for internal administration. It is well tolerated by children and rarely causes toxic symptoms.

Purgative drugs are infrequently indicated in childhood. The two most universally used are castor oil and calomel.

Agar agar, an almost odorless and tasteless mass, is employed to overcome constipation by increasing peristalsis through its "roughage" effect in the intestines. It takes up a great amount of water and swells considerably. It is not digested nor does it undergo any fermentative changes in the intestine. It acts purely in a mechanical way. In its granular form it may be given with cereals. It may also be prescribed in the form of wafers.

Calomel should always be given upon an empty stomach in order to secure the best results. It may be given alone, best followed with a simple purgative, or in combination with phenolphthalein. Its action is both purgative and diuretic, but over-action may result in a production of stomatitis and irritation of the kidneys. In small, repeated doses it has a quieting effect upon the stomach and at times will relieve vomiting and reversed peristalsis. If given in powder form the calomel should be placed in a spoon and water put over it, and then administered. Should the patient become unruly and endeavor to blow the medicine away, some of the water will be blown out but the powder will remain.

Cascara is a mild laxative prescribed for regulating the bowels. However, its use in this respect is abused.

Castor oil, the most popular purgative, causes a rapid and complete emptying of the gastro-intestinal tract. It is indicated in indigestion, particularly when accompanied with diarrhea. The dose should be large, so as to produce prompt results. It is more readily administered if given with a little orange juice, lemon juice, or brandy. If it causes griping a few drops of the deodorized tincture of opium or paregoric may be added to it. Castor oil is sometimes given with equal parts of syrup of rhubarb.

Liquid petrolatum, also known as Russian oil or liquid paraffin, is a more or less refined petroleum. It is used in chronic constipation. It is neither digested nor absorbed but is employed for its mechanical effect.

Magnesium salts are among the most generally used purgatives. The most common preparations are the citrate of magnesia, milk of magnesia, magnesium oxid (calcined magnesia) and magnesium sulphate (epsom salts).

Magnesium citrate, spoken of as "a purgative lemonade," is a mild purgative. It is particularly useful when vomiting is present, for which condition it may be administered repeatedly in small doses. The addition of a little orange juice may render it more palatable to some patients.

Milk of magnesia is especially well borne by infants and children. To infants it may be administered in the bottle feeding. It may also be employed as an antacid in the feedings. In combination with syrup of rhubarb it is particularly efficient as a mild purgative. It is more palatable when given with a little orange juice. Milk of magnesia, when used alone, is best given at bedtime.

Magnesium oxid (calcined magnesia) is the least used of the magnesium salts owing to its excessive bulk.

Magnesium sulphate is more useful with older children. It is unpleasant to take, although it may be rendered more palatable by being well diluted with water. It is more effective if given in concentrated form when there is much dropsy. As a hydrogogue it is especially advantageous in cardiac and renal dropsy. It should not be given to emaciated or weakened individuals. It is not well tolerated when there is vomiting.

Rhubarb, in the form of the syrup, is a popular purgative and may be given alone or in combination, particularly with milk of magnesia or castor oil.

Senna, as the syrup, acts very much like rhubarb.

Sodium phosphate is useful with older children to regulate the bowels by its hepatic stimulant effect. It is indicated in cases of constipation due to diminished biliary secretion, and in cases of chronic duodenitis and catarrhal jaundice.

Quinin is particularly well borne by children to whom relatively larger doses are given than to adults. It is a specific for malaria. It may be administered with syrup of yerba santa or syrup of chocolate in order to disguise its taste. The use of bromids will often lessen the nervous symptoms sometimes caused by the administration of quinin.

Salicylates and salicylic acid are advantageous for the relief of pain, particularly those of rheumatic origin. The drugs are well borne. The most popular preparations are the salicylate of soda and acetylsalicylic acid (aspirin). These drugs are anodyne, antipyretic, diaphoretic, and also diuretic. The bowels should be kept open during the administration of these drugs. Alkalies should not be prescribed with aspirin. Certain individuals have an idiosyncrasy to the drug, in which instance it should not be prescribed. It is not to be used in cases of acute nephritis. In all painful conditions, however, and especially those of rheumatic origin, this drug is indicated. The bromin sodium salicylate or diuretin is a diuretic with a specific action upon the renal epithelium. As it is slightly irritating to the kidneys it should not be employed in acute kidney disease. It is indicated in cardiac disease with edema.

Salvarsan and neosalvarsan are used in the treatment of syphilis and are especially indicated in congenital syphilis. They have been employed in other diseases with questionable results. They are now almost exclusively given intravenously. The toxicity of the drugs should always be kept in mind, though the possibility of their toxic effect is insignificant when compared with the ravages of the disease for which they are given. The drugs may cause irritation of the kidneys and liver, hence, the function of these organs should be carefully watched.

Sodium benzoate is used to render the urine acid when treating pyelitis, so that the administration of hexamethylenamin may be efficient.

Strychnin is as useful in childhood as in later life and may be given in relatively large doses. Care must be exercised not to over-stimulate the patient, which will result in nervous irritability. The drug is usually given by mouth but in emergencies it may be given hypodermically. It is a good tonic in many of its preparations, and is especially advocated as a stimulant for the appetite. Tincture of nux vomica is the most common form in which strychnin is given to children. Compound syrup of hypophosphite is another good tonic containing strychnin.

Sweet spirits of niter is at most universally employed for infants and children, and is to be found in almost every household medicine chest. It is employed as an antipyretic, diaphoretic and mild diuretic. It should be given well diluted as it is likely to cause vomiting if the stomach is irritable.

ORGANOTHERAPY

Of the products derived from the various glands of internal secretions only those of the thyroid, suprarenal, and pituitary glands are of proved worth.

Thyroid extract is a specific in the treatment of hypothyroidism, myxedema and cretinism. It is given by mouth.

Epinephrin, the active principle of the suprarenal gland, is employed usually in solution in the form of the chlorid in the strength of 1 : 1000. Its therapeutic value lies in its stimulating cardiac effect and in its vasoconstrictor effect in surgery. It is useful in raising the blood pressure, and is also valuable in asthma and urticaria. For its systemic effect it is given by needle. Its effect also may be obtained, though less reliably, when the solution is dropped beneath the tongue.

Liquor hypophysis (pituitrin) is obtained from the posterior lobe of the pituitary gland and contains the active principle of this gland. It is employed for its therapeutic effect upon unstriped muscular tissue. Its therapeutic field in children is very limited. It is indicated in shock and may be of service in tympanites and in retention of urine. It is usually given by needle.

Acromegaly and gigantism are believed to be due to hypersecretion of the pituitary gland and are usually associated with tumors and hypertrophy of the gland. Products of the pituitary gland are given in certain cases of infantilism.

The parathyroids have been held accountable by some authorities for the condition of tetany. Other observers, however, hold that disturbed calcium metabolism is responsible for the condition. It may be that the parathyroids have certain controlling influences upon calcium metabolism.

Enlargement of the thymus gland is usually associated with the condition known as status lymphaticus. The therapeutic value of the substance of this gland has not yet been demonstrated.

Much clinical experience is being obtained in the use of the products of the other glands of internal secretion, many of which are being extensively experimented with in animals, but it must be stated that as yet their therapeutic value has not been definitely proved.

DRUGS CLASSIFIED ACCORDING TO THEIR ACTION

The most useful drugs for the treatment of the diseases of infancy and childhood my be grouped according to their principal actions as follows:

Alteratives
  Arsenic
  Cod-liver oil
  Iodin
  Mercury
Anesthetics
  Chloroform
  Ether } General
  Ethyl chlorid
  Nitrous oxide gas
  Cocain
  Ethyl chlorid } Local
  Novocain
Anodyne
  Acetylsalicylic acid
  { Acetanilid
  Coal-tar products { Antipyrin
  { Phenacetin
  Codein
  Morphin
Anthelmintics
  Aspidium
  Chenopodium oil
  Santonin
  Pelletierin (Tanret)
  Thymol
Antipyretics
  Acetanilid
  Antipyrin
  Phenacetin
  Quinin (Malaria)
  Salicylates (Acetylsalicylic acid)
  Spiritus ætheris nitrosi (Sweet spirits of niter)
Antiscorbutics
  Citric acid

- Orange juice
- Lemon juice
- Tomato juice

Antispasmodics
- Asafetida
- Belladonna
- Bromids
- Camphor
- Chloroform
- Ether
- Morphin

Astringents
- Alum
- Bismuth
- Catechu
- Kino
- Tannic acid
- Silver
- Zinc

Carminatives
- Asafetida
- Cardamom
- Cinnamon
- Peppermint
- Spearmint

Counterirritants
- Cantharides
- Chloroform
- Mustard
- Turpentine

Diaphoretics
- Sweet spirits of niter

Diuretics
- Ammonium salts
- Caffein
- Digitalis
- Hexamethylenamin
- Lithium salts
- Potassium salts
- Sodium salts
- Spiritus ætheris nitrosi
- Theobromin sodiosalicylate

Emetics
- Ipecac
- Apomorphin hydrochlorid
- Mustard

Expectorants
- Ammonium carbonate

- Ammonium chlorid
- Compound mixture of glycyrrhiza
- Creosote
- Ipecac (Including wine of ipecac)
- Potassium iodid
- Scilla (Squill)
- Tolu (Syrup)
- Antimony

Hypnotics and narcotics

- Bromids
- Chloral hydrate
- Codein
- Morphin
- Opium
- Trional

Laxatives and purgatives

- Agar agar
- Calomel
- Cascara
- Castor oil
- Jalap (Compound powder)
- Jalap (Compound tincture)
- Liquid petroleum
- Magnesium citrate
- Magnesium milk
- Magnesium sulphate
- Olive oil
- Potassium and sodium tartrate (Rochelle salt)
- Rhubarb
- Sodium sulphate (Glauber's salt)

Sedatives

- Acetanilid
- Acetylsalicylic acid
- Antipyrin
- Belladonna
- Bromids
- Chloral
- Phenacetin
- Salicylates
- Trional

Stimulants

- Adrenalin
- Alcohol
- Ammonium
- Atropin
- Caffein
- Camphor
- Digitalis

- Strophanthus
- Strychnin

Tonics

- Arsenic
- Cod-liver oil
- Hypophosphites (compound syrup)
- Iron
- Malt preparations
- Nux vomica

DRUG DOSAGE

Several factors must be considered in determining the dosage of a drug: (1) The route by which it is to be administered; (2) the character of the drug; (3) the age and weight of the patient.

1. Drugs may be employed (*a*) externally, (*b*) hypodermically, (*c*) perorally, and (*d*) rectally. Certain drugs may also be given intravenously, but these are only a few and may be given special consideration in connection with the diseases in which they are indicated.

2. Those drugs which are toxic and those which are not well borne by children must be given with especial caution, and in doses much smaller than determined by the usual methods of calculation. A marked susceptibility of the child to a special drug must not be overlooked.

3. The age of the patient should influence the size of the dose, but as many of the patients who require medication are below normal size for their age, the dosage if calculated from this factor alone may be too large.

4. For this reason the determination of dosage on the basis of the weight of the child is a better guide.

The route by which drugs are administered is an influencing factor. Considering the dose as calculated by any of the following methods for internal (peroral) administration, it might be said safely that half that dose may be given hypodermically and double the dose by rectum. Of course, not all the drugs may be given hypodermically and not all the drugs are of service when given by rectum. When drugs are used externally they may be prescribed in larger dosage.

Various methods for the determination of the dosage of drugs have been devised. Young's method is to divide the age (in years) of the patient by the age plus 12, the resulting fraction giving the size of the dose for the child as compared with that for the adult. Clarke divides the weight (in pounds) of the patient by 150 (being the average weight of the adult) and the resulting fraction is his guide for the dose to be given. Cowling uses the age at the next birthday as the numerator and 24 as the denominator, and this fraction is the size of the dose to be given.

These methods do not take into consideration both the size and the age of the patient, and as all children vary in these two factors, the writer employs the ordinary calculation of Young, but modifies the latter's rule by using the age to which the patient's weight corresponds as the numerator and that age plus 12 as the denominator to determine the fraction of the adult dose to be prescribed. One-twelfth of the dose at one year is given for each month of life during the first year. Besides the size of the dose, other factors should be kept in mind when prescribing, namely, the method of employment; the toxicity of the drug; the idiosyncrasy of the patient; the time of administration with relation to meals and sleep; the frequency of administration; the physiologic action of the drug; preparation of the drug, and the condition of disease. The intelligence of the parents and attendants of the patient should not be overlooked.

ANESTHETICS

The methods of administration of anesthetics to children are the same as those employed in the adult. There are several differences, however, which may influence the child's ability to take the anesthetic.

1. The young individual is relatively free from organic disease particularly of the heart and lungs. (2) The lung area is proportionately greater and hence there is a relatively larger lung capacity. (3) The younger the child the less unstable is its nervous control.

The relative freedom from organic disease, and particularly of the heart and lungs, may be accountable for the fewer fatal accidents after anesthesia in childhood as compared with later life. At the same time it must be remembered that there is no special resistance against the lethal action of the various anesthetic drugs during the early period of life.

The anesthetics may be divided into two groups, those for general and those for local anesthesia. The former include chloroform, ether, ethyl chlorid, nitrous oxid and their various combinations. The latter are ethyl chlorid, cocain, covocain and beta-eucain (etc.).

General Anesthetics

Certain variations in the reflexes are observed during the administration of anesthetics to young patients as compared with adults, for example: The corneal reflex is more unreliable in that it may be present in one eye and absent in the other; or it may be present with no other signs of sensation, or it may be absent and the patient show signs of sensation. Spasm of the glottis is more readily produced than in the adult, particularly accompanying painful surgical procedures. Other reflexes are less active, probably owing to lack of inhibition over certain functions.

Fright in the child will frequently cause a lack of coöperation and a resistance to the taking of the anesthetic with the resulting deeper breathing. This should be taken into consideration in giving the drug so that it will not be given in too great quantity, nor too fast. Finally, it should be borne in mind that the child is more susceptible to shock than is the adult.

Ether is the anesthetic now generally employed and should be the one of choice unless there are contra-indications to its use, or other reasons for not giving it. Ether possesses many advantages over the other anesthetics.

As compared with chloroform ether is:

1. Quicker in action.
2. It causes less resistance which is of shorter duration.
3. It is less depressing.
4. It has a stimulating effect upon the circulatory system. The pulse remains strong throughout.
5. The respiratory system is less disturbed, the respirations being deeper and active. The face keeps a good color.
6. The patient is more promptly anesthetized and the anesthesia is more easily graduated and controlled.
7. If the patient should show a tendency to the return of consciousness the anesthesia is more speedily and safely re-induced.
8. There is less tendency to syncope.
9. The overdose risk is very slight.
10. There is less disturbance following the anesthesia.

Its disadvantages are:

1. The necessity for special apparatus.
2. Its unpleasantness when given alone (overcome by giving nitrous oxid at onset).
3. Tendency to cause excessive secretion of mucus.

Ether is to be preferred when the anesthetist is inexperienced, in conditions of prolonged anesthesia, and in cardiac disease with imperfect compensation. It is the anesthetic of choice in tonsil and adenoid operations. Its contra-indications are bronchitis, pneumonia, pleurisy, conditions causing obstructive respiration, and nephritis or conditions predisposing thereto, as for example: Scarlet fever.

Chloroform is not now considered as safe as was formerly believed. Children possess no unusual tolerence for chloroform nor any special power of resistance to its lethal action.

Its advantages are:

1. The simplicity of apparatus.
2. Its pleasant flavor.
3. The small quantity necessary for anesthesia.
4. The almost complete absence of bronchial irritation.

Its disadvantages are:

1. It is slow in action.
2. The resistance is marked and prolonged.
3. It is depressing, causes pallor, feeble pulse, dilated pupils.
4. It causes shallow respiration.
5. There is difficulty in estimating and controlling the anesthesia.
6. There is difficulty in re-inducing anesthesia when signs of recovery become manifest.
7. An overdose is easy.
8. The disturbances following anesthesia are considerable.

Chloroform should be used as an anesthetic in those conditions in which ether is contra-indicated, as for example: In respiratory disease; in conditions of obstructive respiration; and in nephritis and its predisposing ailments. The desired effect should be produced with the minimum amount of the drug and a maximum amount of air. In a struggling child the administration of chloroform becomes more hazardous. Chloroform is contra-indicated in the lymphatic diathesis, in operation on tonsils and adenoids, and in conditions of weakness and prostration.

Nitrous oxid is not well borne by children, particularly under two years. It may be given when anesthesia of a few moments only is desired. In older children nitrous oxid produces such a prompt anesthesia and its effect is so lasting that special caution should be exercised in its administration. It should not be employed for prolonged anesthesia and never when there is any disturbance in breathing. Nitrous oxid is sometimes given as a preliminary to ether and it facilitates the administration of the latter.

Ethyl chloride is more generally used for short anesthesia, having in a great measure supplanted nitrous oxid. It requires less apparatus and while the usual inhaler is to be preferred, ethyl chlorid may be given by spraying it on a handkerchief or lint. It should never be given after unconsciousness has already been induced. Ethyl chlorid produces less muscular disturbance than does nitrous oxid but it is probably less safe and after-effects (headache and nausea) are more common. Ethyl chlorid may be given in order to produce rapid unconsciousness either before ether or chloroform anesthesia.

Local Anesthetics

Much progress has been made within recent years in the administration of drugs for the production of local anesthesia. The field for this type of anesthesia in children is rather limited at this time. Undoubtedly some of the advantages in the employment of local anesthesia are the absence of the necessity for the preparation of the patients and the absence of subsequent general discomfort. Local anesthesia should not be attempted in patients who are disinclined to coöperate, nor in those whose unstable nervous system will cause them to suffer nervous shock. Such nervous shocks, have, at times, created profound impressions, the effects of which have been prolonged, lasting and injurious. For the foregoing reasons local anesthesia is frequently contra-indicated in children, in whom the combination of opiates with local anesthesia may be sufficient. If, however, the latter measure fails, general anesthesia must be resorted to.

Local absence of pain may be produced by spinal injection, spray, and infiltration. The spinal route has not been satisfactory in the child. In this method cocain and novocain and tropacocain are employed, the latter being the most popular because less unfavorable results have been reported from its use.

The spray method, that of using ethyl chlorid to produce anesthesia by freezing the skin surface, serves a decided purpose and is of especial value.

The infiltration method has been so perfected that with this aid major abdominal operations are now performed regularly by many operators, and with as much success in the child who coöperates as in the adult. The drugs used in the production of local anesthesia by infiltration are cocain, novocain, beta-eucain, etc.

Cocain is generally employed and is most efficient. It produces no pain or inflammation when injected. However, it has a high toxicity.

Novocain is not quite so powerful in its anesthetic action as cocain, but its effect is more durable. Like cocain it is not at all irritating to the tissues. It is probably the least toxic, the safest, most reliable and satisfactory of all the local anesthetic agents.

Beta-eucain is quite useful for the production of local anesthesia. It is slower in its action than either cocain or novocain and nearly as effective. It is less toxic than cocain being nearly as weak in its toxicity as novocain. Its injection into the tissues is accompanied by a sharp pain that lasts for several minutes.

The other local anesthetics possess no advantages over those already mentioned, but really have many disadvantages. Among these drugs may be mentioned stovain, alypin, and tropacocain. The success of this method of anesthesia rests in a great measure in the ability to secure and retain the confidence of the child. The differentiation between pain and sensation on the part of the patient and a knowledge regarding the administration of the anesthetic are the two prime factors in the successful outcome of the procedure.

KEY TO TABLE OF DRUGS

The doses in the table are computed in nearly every instance upon the basis of the adult doses as given in the United States Pharmacopeia.

Abbreviations in the column of solubilities are:

alm.—almost
dec.—decomposed
ins.—insoluble
p.—partially
r.—readily
s.—soluble
sl.—slightly
sp.—sparingly
v.—very

It is advisable as a rule in prescribing drugs to begin with the smaller doses appearing in this table unless otherwise indicated.

TABLE OF DRUGS AND

Name	*Solubility of one part*		*Adult dose*
	In water At 77°F. At 25°C.	*In alcohol At 77°F. At 25°C.*	
Acetanilid	190	3.4	3–8 gr. 0.2–0.5 gm.
Acetphenetidin (Phenacetin)	1310	15	5–15 gr. 0.32–1.0 gm.
Acid Acetylsalicylic (Aspirin)	sl. s.	s.	5–15 gr. 0.32–1.0 gm.
Acid Arsenious	30–100	sp.	1/40–1/20 gr. 0.0016–0.003 gm.
Acid Benzoic	275	2.3	10–30 gr. 0.65–2.0 gm.
Acid Citric	0.5	1.8	5–30 gr. 0.32–2.0 gm.
Acid Gallic	87	4.6	5–20 gr. 0.32–1.3 gm.
Bismuth Subgallate	ins.	ins.	7–30 gr. 0.45–2.0 gm.
Acid Hydrochloric dil.	....	....	10–60 m. 0.6–3.75 mil.
Acid Phosphoric dil.	....	....	15–60 m. 0.9–3.75 mil.
Acid Salicylic	460	2.7	5–20 gr. 0.32–1.3 gm.
Aspirin (Acid Acetylsalicylic)	sl. s.	s.	5–15 gr. 0.32–1.0 gm.
Bismuth Subsalicylate	alm. ins.	....	10–20 gr. 0.65–1.3 gm.
Methyl Salicylate	sp. s.	6	5–20 m. 0.3–1.3 mil.
Salol (Phenyl Salicylate)	6670	6	5–15 gr. 0.32–1.0 gm.
Sodium Salicylate	0.9	9.2	10–30 gr. 0.65–2.0 gm.
Theobromin (Sodiosalicylate)	1	sp.	5–10 gr. 0.32–0.65 gm.
Acid Tannic	vs.	vs.	3–20 gr. 0.20–1.3 gm.
Tannalbin (Tannin Albuminate)	ins.	ins.	15–30 gr. 1.0–2.0 gm.
Tannigen (Tannyl Acetate)	ins.	s.	10–15 gr. 0.65–1.0 gm.
Acid Tartaric	0.75	3.3	5–30 gr. 0.32–2.0 gm.
Potassium and Antimony Tartrate (Tartar Emetic) (as diaphoretic and expectorant).	12	ins.	1/20–1/6 gr. 0.003–0.01 gm.
Potassium and Antimony Tartrate (as emetic)	12	ins.	½ gr. 0.032 gm.
Potassium Bitartrate (Cream of Tartar) (aperient)	155	v. sp.	1–2 dr. 3.9–7.7 gm.
Potassium Bitartrate (cathartic)	155	v. sp.	½–1 oz. 15.5–31 gm.
Potassium and Sodium Tartrate (Rochelle salt)	0.9	alm. ins.	½–1 oz. 15.5–3.0 gm.
Aconite Root, tincture	....	....	3–10 m. 0.2–0.6 mil.
Adrenalin (Epinephrin) (intramuscular)	sp.	alm. ins.	1/200–3/200 gr. 0.0003–0.0009 gm.
Adrenalin. Sol. (1/1000 by needle for systemic effect) (not subcutaneous)	....	....	5–20 m. 0.3–1.3 mil.
Agar Agar	ins.	ins.	2–4 dr. 7.7–15.5 gm.
Alcohol			
Brandy	....	....	1–4 dr. 3.9–15.5 mil.
Champagne	....	......	4 dr.–2 oz. 15.5–62 mil.
Gin	....	....	1–4 dr. 3.9–15.5 mil.
Port Wine	....	....	3 dr.–1½ oz. 11.7–46.5 mil.
Sherry Wine	....	....	3 dr.–1½ oz. 11.7–45.5 mil.
Whisky	....	....	1–4 dr. 3.9–15.5 mil.
Aloes (not advised in the treatment of children)			
Aloin	65	10.75	1/6–1 gr. 0.01–0.065 gm.
Alum (not advised in the treatment of children)			

DOSAGE FOR INTERNAL USE

Six months or 15 *lb.* 6.8 *kg.*	*One year or* 22 *lb.* 10 *kg.*	18 *months or* 25 *lb.* 11.35 *kg.*	*Two years or* 28 *lb.* 12.70 *kg.*	*Three years or* 32 *lb.* 14.64 *kg.*
1/10–3/10 gr.	⅕–⅗ gr.	3/10–1 gr.	⅖–1 gr.	⅗–1⅗ gr.
0.006–.018 gm.	0.013–0.04 gm.	0.019–0.065 gm.	0.026–.068 gm.	0.04–0.1 gm.
⅙–½ gr.	⅓–1 gr.	7/10–1⅔ gr.	5/7–2 gr.	1–3 gr.
0.01–0.03 gm.	0.021–0.06 gm.	0.042–0.1 gm.	0.043–0.128 gm.	0.065–0.2 gm.
⅕–⅗ gr.	⅓–1 gr.	½–1½ gr.	¾–2 gr.	1–3 gr.
0.013–0.039 gm.	0.021–0.065 gm.	0.03–0.09 gm.	0.048–0.12 gm.	0.065–0.18 gm.
1/1000–1/500 gr.	1/500–1/250 gr.	1/300–1/50 gr.	1/250–1/140 gr.	1/200–1/100 gr.
0.00001–0.00012 gm.	0.00012–0.00024 gm.	0.0002–0.0004 gm.	0.00024–0.00042 gm.	0.0003–0.0006 gm.
½–1 gr.	¾-2 gr.	1–3 gr.	1½–4 gr.	2–6 gr.
0.03–0.065 gm.	0.048–0.12 gm.	0.065–0.18 gm.	0.09–0.26 gm.	0.12–0.36 gm.
⅕-1 gr.	⅓–2 gr.	½–3 gr.	¾–4 gr.	1–6 gr.
0.013–0.065 gm.	0.02–0.12 gm.	0.03–0.18 gm.	0.048–0.26 gm.	0.065–0.36 gm.
⅕–¾ gr.	⅓–1½ gr.	½–2 gr.	¾–3 gr.	1–4 gr.
0.012–0.048 gm.	0.021–0.09 gm.	0.03–0.12 gm.	0.048–0.18 gm.	0.065–0.26 gm.
¼-1 gr.	½–2 gr.	¾–3 gr.	1–4 gr.	1½–6 gr.
0 016–0.065 gm.	0.03–0.12 gm.	0.048–0.18	0.065–0.26 gm.	0.09–0.36 gm.
⅖–2 m.	½–4 m.	1–6 m.	1½–8 m.	2–12 m.
0.026–0.12 mil.	0.03–0.26 mil.	0.065–0.36 mil.	0.09–0.48 mil.	0.12–0.72 mil.
½–2 m.	1–4 m.	1½–6 m.	2–8 m.	3–12 m.
0.03–0.12 mil.	0.065–0.26 mil.	0.09–0.36 mil.	0.12–0.48 mil.	0.18–0.72 mil.
⅕–¾ gr.	⅓–1½ gr.	½–2 gr.	¾–3 gr.	1–4 gr.
0.013–0.048 gm.	0.02–0.09 gm.	0.03–0.12 gm.	0.048–0.18 gm.	0.065–0.26 gm.
⅕–⅗ gr.	⅓–1 gr.	½–1½ gr.	¾–2 gr.	1–3 gr.
0.013–0.039 gm.	0.021–0.065 gm.	0.03–0.09 gm.	0.048–0.18 gm.	0.065–0.18 gm.
½–¾ gr.	¾-1½ gr.	1–2 gr.	1½–3 gr.	2–4 gr.
0.03–0 048 gm.	0.048–0.09 gm.	0.06–0.18 gm.	0.09–0.18 gm.	0.18–0.26 gm.
⅕–¾ m.	⅓–1½ m.	½–2 m.	¾–3 m.	1–4 m.
0.012–0.048 mil.	0.021-0.09 mil.	0.03–0.12 mil.	0.048–0.18 mil.	0.065–0.26 mil.
⅕–½ gr.	⅓–1 gr.	½–2 gr.	¾–2 gr.	1–3 gr.
0.013–0.03 gm.	0.02–0.065 gm.	0.03–0.12 gm.	0.048–0.12 gm.	0.065–0.18 gm.
⅖–1 gr.	¾–2¼ gr.	1–3 gr.	1½–4¼ gr.	2–6 gr.
0.026–0.065 gm.	0.048–0.136 gm.	0.065–0.18 gm.	0.09–0.278 gm.	0.12–0.36 gm.
⅕–⅖ gr.	⅓–¾ gr.	½–1 gr.	¾–1½ gr.	1–2 gr.
0.013–0.026 gm.	0.02–0.048 gm.	0.03–0.065 gm.	0.048–0.09 gm.	0.065–0.13 gm.
½–¾ gr.	1–1½ gr.	1¾–2¼ gr.	2–3 gr.	3–4 gr.
0.03–0.048 gm.	0.065–0.09 gm.	0.11–0.136 gm.	0.03–0.18 gm.	0.18–0.26 gm.
½–1 gr.	1–2¼ gr.	1¾–3½ gr.	2–4¼ gr.	3–6 gr.
0.03–0.065 gm.	0.065–0.136 gm.	0.11–0.21 gm.	0.03–0.276 gm.	0.18–0.36 gm.
⅖–½ gr.	¾–1 gr.	1–1¾ gr.	1½–2 gr.	2–3 gr.
0.026–0.03 gm.	0.048–0.065 gm.	0.065–0.11 gm.	0.09–0.03 gm.	0.03–0.18 gm.
⅕–1 gr.	⅓–2¼ gr.	½–3¾ gr.	¾–4 gr.	1–6 gr.
0.013–0.065 gm.	0.02–0.136 gm.	0.03–0.228 gm.	0.048–0.26 gm.	0.065–0.36 gm.
1/500–1/150 gr.	1/250–1/75 gr.	1/150–1/50 gr.	1/140–1/40 gr.	1/100–1/30 gr.
0.00012–0.0004 gm.	0.00024–0.0008 gm.	0.0004–0.0012 gm.	0.00043–0.0015 gm.	0.0006–0.002 gm.
1/50 gr.	1/25 gr.	1/16 gr.	1/14 gr.	1/10 gr.
0.0012 gm.	0.0024 gm.	0.0037 gm.	0.0035 gm.	0.006 gm.
2½–5 gr.	5–10 gr.	7–15 gr.	9–18 gr.	12–25 gr.
0.15–0.3 gm.	0.3–0.6 gm.	0.42–1.0 gm.	0.54–1.08 gm.	0.72–1.5 gm.
7½–15 gr.	17–35 gr.	25–45 gr.	35–70 gr.	80–96 gr.
0.45–1.0 gm.	1.02–2.1 gm.	1.5–2.7 gm.	2.1–4.2 gm.	4.8–5.76 or 6 gm.
7½–15 gr.	17–30 gr.	25–45 gr.	35–70 gr.	40–90 gr.
0.45–1.0 gm.	1.02–2 gm.	1.5–2.7 gm.	2.1–4.2 gm.	2.4–6.0 gm.
1/10–⅗ m.	⅕–¾ m.	⅓–1 m.	½–1½ m.	⅔–2 m.
0.006–0 026 mil.	0.013–0.048 mil.	0.02–0.065 mil.	0.03–0.09 mil.	0.04–0.12 mil.
1/5000–1/1750 gr.	1/2500–1/875 gr.	1/1600–1/500 gr.	1/1400–1/450 gr.	1/1000–1/300 gr.
0.000012–0.000036 gm.	0.000024–0.000072 gm.	0.000037–0.000091 gm.	0.000043–0.00012 gm.	0.00006–0.0018 gm.
⅕–¾ m.	⅓–1½ m.	½–2 m.	¾–3 m.	1–4 m.
0.013–0.048 mil.	0.02–0.09 mil.	0.03–0.12 mil.	0.048–0.18 mil.	0.065–0.26 mil.
5–10 gr.	10–20 gr.	15–30 gr.	20–40 gr.	25–50 gr.
0.3–0.65 gm.	0.065–1.3 gm.	1.0–2.0 gm.	1.3–2.4 gm.	1.5–3.0 gm.
3–10 m.	5–20 m.	8–30 m.	10–40 m.	12–50 m.
0.18–0.65 mil.	0.32–1.3 mil.	0.48–2.0 mil.	0.65–2.5 mil.	0.72–3.0 mil.
10–40 m.	20–80 m.	30–120 m.	40–60 m.	50–200 m.
0.65–2.5 mil.	1.3–5.0 mil.	2.0–8.0 mil.	2.6–10.6 mil.	3.25–13.0 mil.
3–10 m.	5–20 m.	8–30 m.	10–40 m.	12–50 m.
0.18–0.65 mil.	0.32–1.3 mil.	0.48–2.0 mil.	0.65–2.5 mil.	0.72–3.0 mil.
9–30 m.	15–60 m.	24–90 m.	30–120 m.	36–150 m.
0.54–2.0 mil.	1.0–4.0 mil.	1.44–6.0 mil.	2.0–8.0 mil.	2.16–10.0 mil.
9–30 m.	15–60 m.	24–90 m.	30–120 m.	36–150 m.
0.54–2.0 mil.	1.0–4.0 mil.	1.44–6.0 mil.	2.0–8.0 mil.	2.16–10.0 mil.
3–10 m.	5–20 m.	8–30 m.	10–40 m.	12–50 m.
0.18–0.65 mil.	0.32–1.3 mil.	0.48–2.0 mil.	0.65–2.5 mil.	0.72–3.0 mil.
1/150–1/26 gr.	1/75–1/13 gr.	1/60–⅛ gr.	1/40–1/7 gr.	1/30–⅕ gr.
0.0004–0.0025 gm.	0.008–0.005 gm.	0.0012–0.008 gm.	0.0015–0.009 gm.	0.002–0.013 gm.

TABLE OF DRUGS AND DOSAGE

Name	*Solubility of one part*		*Adult dose*
	In water At 77°F. At 25°C.	*In alcohol At 77°F. At 25°C.*	
Ammonia			
Ammonium acetate, Sol. (Spts. Mindererus)	....	....	½–1 oz. 15–30 mil.
Ammonium, Aromatic spts. of	....	....	30–60 m. 1.8–3.75 mil.
Ammonium Bromid	1.3	12	10–30 gr. 0.6–1.9 gm.
Ammonium Chlorid	2.6	100	5–10 gr. 0.32–0.65 gm.
Ammonium carbonate	4	dec.	5–10 gr. 0.32–0.65 gm.
Anise oil	....	....	3–8 m. 0.2–0.5 mil.
Anise seed	ins.	ins.	8–15 gr. 0.5–1.0 gm.
Antimony and Potassium Tartrate (Tartar emetic)	12	ins.	1/20–1/6 gr. 0.003–0.01 gm.
Antipyrin (Phenazone)	v. s.	1.3	3–15 gr. 0.2–1.0 gm.
Antitoxin, Diphtheria			
Immunization	....	....	1500 units
Pharyngeal	....	....	20,000 units
Laryngeal	....	....	40,000 units
Apomorphin (not advised in the treatment of children)			
Arsenious acid (Arsenic Trioxide)	30–100	sp.	1/40–1/20 gr. 0.0016–0.003 gm.
Arsenitis, Liq. Potas	....	....	3–5 m. 0.2–0.3 mil.
Asafœtida, emulsion of (milk of Asafœtida)	....	....	½–1 oz. 15–30 mil.
Aspidium Oleoresin	....	....	½–1 fl. dr. 1.8–3.75 mil.
Aspirin (Acid Acetylsalicylic)	sl. s	s.	5–15 gr. 0.32–1.0 gm.
Atropin	455	2	1/100–1/60 gr. 0.0003–0.0013 gm.
Barium Sulphate (In roentgenography) (see External Drugs)			
Basham's Mixture (Liq. Ferri et Ammonia Acetate)	....	....	2–4 dr. 7.5–15 mil.
Belladonna, tincture of	....	....	5–15 m. 0.3–0.9 mil.
Benzoic Acid	275	2.3	10–30 gr. 0.65–2.0 gm.
Benzyl Benzoate (20 per cent. alcohol sol.)	....	....	20–30 m. 1.3–1.8 mil.
Beta naphthol	1000	0.8	2–5 gr. 0.13–0.32 gm.
Bichlorid of Mercury	13.5	3.8	1/40–1/8 gr. 0.0015–0.008 gm.
Bismuth subcarbonate	ins.	ins.	10–30 gr. 0.65–2.0 gm.
Bismuth subcarbonate (see External Drugs) (in roentgenography)			
Bismuth subgallate	ins.	ins.	7–30 gr. 0.45–2.0 gm.
Bismuth subnitrate	alm. ins.	alm. ins.	5–30 gr. 0.32–2.0 gm.
Bismuth subsalicylate	alm. ins.	....	10–20 gr. 0.65–1.3 gm.
Blaud's Pills	....	....	5–20 gr. (2–6 pills) 0.32–1.3 gm.
Borax (Sodium borate)	15	ins.	10–20 gr. 0.65–1.3 gm.
Brandy (see Alcohol)			
Bromid, Ammonium	1.3	12	10–30 gr. 0.65–1.9 gm.
Bromid, Potassium	1.5	250	10–45 gr. 0.65–3.0 gm.
Bromid, Sodium	1.1	16	10–60 gr. 0.65–3.9 gm.
Bromid, Strontium	0.35	r. s.	15–30 gr. 1.0–2.0 gm.
Bromoform	v. sp.	v. s.	1–5 m. 0.06–0.3 mil.
Brown Mixture (Mistura Glycyrrhizae Composite)	....	....	½–1 oz. 15–30 mil.
Buchu, fluid extract	....	....	30–60 m. 1.8–3.75 mil.

FOR INTERNAL USE (*Continued*)

Six months or 15 *lb.* 6.8 *kg.*	*One year or* 22 *lb.* 10 *kg.*	18 *months or* 25 *lb.* 11.35 *kg.*	*Two years or* 28 *lb.* 12.70 *kg.*	*Three years or* 32 *lb.* 14.64 *kg.*
7½–15 m.	17–35 m.	25–45 m.	35–70 m.	80–90 m.
0.45–1 mil.	1.02–2.1 mil.	1.5–2.7 mil.	2.1–4.2 mil.	4.8–5.4 mil
1–2 m.	2–4 m.	3–6 m.	4–8½ m.	6–12 m.
0.06–0.12 mil.	0.12–0.26 mil.	0.18–0.36 mil.	0.26–0.48 mil.	0.36–0.72 mil.
½–1 gr.	¾–2½ gr.	1–3½ gr.	1½–4½ gr.	2–6 gr.
0.03–1.65 gm.	0.048–0.15 gm.	0.065–0.21 gm.	0.09–0.29 gm.	0.12–0.36 gm.
⅕–1 gr.	⅓–2½ gr.	½–3½ gr.	¾–4½ gr.	1–6 gr.
0.013–0.065 gm.	0.02–0.15 gm.	0.03–0.21 gm.	0.048–0.29 gm.	0.065–0.36 gm.
⅕–1 gr.	⅓–2½ gr.	½–3½ gr.	¾–4½ gr.	1–6 gr.
0.013–0.065 gm.	0.02–0.15 gm.	0.03–0.21 gm.	0.048–0.29 gm.	0.065–0.36 gm.
1/10–⅓ m.	⅕–⅔ m.	⅓–1 m.	½–1¼ m.	⅔–1½ m.
0.006–0.02 mil.	0.013–0.04 mil.	0.02–0.065 mil.	0.03–0.08 mil.	0.04–0.09 mil.
⅓–½ gr.	⅔–1 gr.	1–1½ gr.	1¼–2 gr.	1½–3 gr.
0.02–0.03 gm.	0.04–0.065 gm.	0.065–0.09 gm.	0.08–0.12 gm.	0.09–0.18 gm.
1/500–1/150 gr.	1/250–1/75 gr.	1/150–1/50 gr.	1/140–1/40 gr.	1/100–1/30 gr.
0.00012–0.0004 gm.	0.00024–0.0008 gm.	0.0004–0.0012 gm.	0.00042–0.0015 gm.	0.0001–0.002 gm.
1/10–½ gr.	⅕–1 gr.	⅓–1½ gr.	½–2 gr.	⅔–3 gr.
0.006–0.03 gm.	0.013–0.065 gm.	0.02–0.09 gm.	0.03–0.12 gm.	0.04–0.18 gm.
700 units	1,000 units	1,000 units	1,500 units	1,500 units
5,000 units	5,000 units	5,000 units	10,000 units	10,000 units
10,000 units	10,000 units	20,000 units	20,000 units	20,000 units
1/1000–1/500 gr.	1/500–1/250 gr.	1/300–1/150 gr.	1/250–1/140 gr.	1/200–1/100 gr.
0.00001–0.00012 gm.	0.00012–0.00024 gm.	0.0002–0.0004 gm.	0.00024–0.0042 gm.	0.0003–0.0006 gm.
1/10–⅕ m.	⅕–⅓ m.	⅓–½ m.	½–¾ m.	⅔–1 m.
0.006–0.013 mil.	0.013–0.02 mil.	0.02–0.03 mil.	0.03–0.048 mil.	0.04–0.065 mil.
7½–15 m.	15–35 m.	25–45 m.	35–70 m.	80–90 m.
0.45–1 mil.	1.0–2.1 mil.	1.5–2.7 mil.	2.1–4.3 mil.	4.8–5.4 mil.
1½–2½ m.	3–5 m.	4½–7½ m.	5–9 m.	6–12 m.
0.09–0.15 mil.	0.18–0.3 mil.	0.29–0.45 mil.	0.3–0.54 mil.	0.36–0.72 mil.
⅕–½ gr.	⅓–1 gr.	1–3½ gr.	1½–4½ gr.	2–6 gr.
0.013–0.03 gm.	0.02–0.065 gm.	0.065–0.21 gm.	0.09–0.29 gm.	0.12–0.36 gm.
1/2500–1/1250 gr.	1/1250–1/600 gr.	1/900–1/450 gr.	1/700–1/350 gr.	1/500–1/250 gr.
0.000025–0.000048 gm.	0.000048–0.0001 gm.	0.000066–0.00013 gm.	0.000085–0.00017 gm.	0.00012–0.00024 gm.
5–10 m.	10–20 m.	15–30 m.	20–40 m.	25–50 m.
0.3–0.65 mil.	0.65–1.3 mil.	1.0–2.0 mil.	1.3–2.4 mil.	1.5–3.0 mil.
⅕–½ m.	⅓–1 m.	½–2 m.	¾–2 m.	1–3 m.
0.013–0.03 mil.	0.02–0.065 mil.	0.03–0.12 mil.	0.048–0.12 mil.	0.065–0.18 mil.
½–1 gr.	¾–2½ gr.	1–3½ gr.	1½–4½ gr.	2–6 gr.
0.03–0.065 gm.	0.048–0.15 gm.	0.065–0.21 gm.	0.09–0.29 gm.	0.12–0.36 gm.
¾–1 m.	1½–2 m.	2–3 m.	3–4 m.	4–6 m.
0.048–0.65 mil.	0.09–0.13 mil.	0.136–0.18 mil.	0.18–0.26 mil.	0.26–0.36 mil.
1/12–⅕ gr.	⅙–⅓ gr.	¼–½ gr.	⅓–¾ gr.	½–1 gr.
0.005–0.013 gm.	0.01–0.02 gm.	0.016–0.03 gm.	0.02–0.048 gm.	0.03–0.065 gm.
1/1000–1/200 gr.	1/500–1/100 gr.	1/300–1/70 gr.	1/250–1/50 gr.	1/200–1/40 gr.
0.00006–0.0003 gm.	0.00012–0.0006 gm.	0.0002–0.00085 gm.	0.00024–0.0012 gm.	0.0003–0.0015 gm.
½–1 gr.	¾–2½ gr.	1–3½ gr.	1½–4½ gr.	2–6 gr.
0.03–0.065 gm.	0.048–0.15 gm.	0.065–0.21 gm.	0.09–0.29 gm.	0.12–0.36 gm.
¼–1 gr.	½–2½ gr.	¾–3½ gr.	1–4½ gr.	1½–6 gr.
0.016–0.065 gm.	0.03–0.15 gm.	0.048–0.21 gm.	0.065–0.29 gm.	0.09–0.36 gm.
⅕–1 gr.	⅓–2½ gr.	½–3½ gr.	¾–4½ gr.	1–6 gr.
0.013–0.065 gm.	0.02–0.15 gm.	0.03–0.21 gm.	0.048–0.29 gm.	0.065–0.36 gm.
½–¾ gr.	¾–1½ gr.	1–2 gr.	1½–3 gr.	2–4 gr.
0.03–0.048 gm.	0.048–0.09 gm.	0.065–0.12 gm.	0.09–0.18 gm.	0.12–0.26 gm.
⅕–¾ gr.	⅓–1½ gr.	½–2 gr.	¾–3 gr.	1–4 gr.
0.013–0.048 gm.	0.02–0.09 gm.	0.03–0.12 gm.	0.048–0.18 gm.	0.065–0.26 gm.
1–¾ gr.	2½–1½ gr.	3½–2 gr.	4½–3 gr.	6–4 gr.
0.065–0.048 gm.	0.15–0.09 gm.	0.21–0.12 gm.	0.29–0.18 gm.	0.36–0.26 gm.
⅖–1 gr.	¾–2 gr.	1–3 gr.	1½–4 gr.	2–6 gr.
0.026–0.065 gm.	0.048–0.12 gm.	0.065–0.18 gm.	0.09–0.26 gm.	0.12–0.36 gm.
⅖–1⅗ gr.	¾–3½ gr.	1–5 gr.	1½–6½ gr.	2–9 gr.
0.026–0.01 gm.	0.048–0.21 gm.	0.065–0.3 gm.	0.09–0.39 gm.	0.12–0.54 gm.
½–2 gr.	¾–4½ gr.	1–6½ gr.	1½–8½ gr.	2–12 gr.
0.03–0.12 gm.	0.048–0.29 gm.	0.065–0.39 gm.	0.09–0.51 gm.	0.12–0.72 gm.
½–1 gr.	1–2¼ gr.	1¾–3½ gr.	2–4½ gr.	3–6 gr.
0.03–0.06 gm.	0.065–0.136 gm.	0.11–0.21 gm.	0.03–0.276 gm.	0.18–0.26 gm.
1/26–⅕ m.	1/13–⅓ m.	⅛–½ m.	⅐–¾ m.	⅕–1 m.
0.002–0.013 mil.	0.004–0.02 mil.	0.008–0.03 mil.	0.009–0.048 mil.	0.013–0.065 mil.
7½–15 m.	15–25 m.	25–45 m.	35–70 m.	80–90 m.
0.45–1 mil.	1.0–2.1 mil.	1.5–2.7 mil.	2.1–4.2 mil.	4.8–5.4 mil.
1–2 m.	2–4 m.	3–6 m.	4–8 m.	6–12 m.
0.06–0.12 mil.	0.12–0.26 mil.	0.18–0.36 mil.	0.26–0.48 mil.	0.36–0.72 mil.

TABLE OF DRUGS AND DOSAGE

Name	*Solubility of one part* *In water* *At 77°F.* *At 25°C.*	 *In alcohol* *At 77°F.* *At 25°C.*	*Adult dose*
Buchu, infusion (see also External Drugs)	....	....	1–2 oz. 30–60 mil.
Caffein	46	66	1–5 gr. 0.065–0.32 gm.
Caffein, citrate of	v. s.	....	3–8 gr. 0.2–0.5 gm.
Caffein Sodiobenzoate (by hypodermic) In solution 1 : 3 water	1.1	30	10–20 m. 0.6–1.3 mil.
Caffein Sodiobenzoate (by mouth)	1.1	30	3–10 gr. 0.2–0.6 gm.
Caffein Sodiosalicylate (by hypodermic) In solution 1: 3 water	v. s.	....	10–20 m. 0.6–1.3 mil.
Caffein Sodiosalicylate (by mouth)	v. s.	....	3–10 gr. 0.2–0.6 gm.
Calcium chlorid	0.62	10	30–60 gr. 2.0–4.0 gm.
Calcium lactate	20	alm. ins.	30–60 gr. 2.0–4.0 gm.
Calcium sulphid (crude)	v. sp.	ins.	¼–1 gr. 0.015–0.065 gm.
Calcium Liquor (see Lime Water)			
Calomel (as alterative)	ins.	ins.	⅙–⅓ gr. 0.01–0.02 gm.
Calomel (as cathartic)	ins.	ins.	2–5 gr. 0.12–0.3 gm.
Camphor Gum	v. sp.	r. s.	2–5 gr. 0.13–0.32 gm.
Camphor in oil (by hypodermic)	....	....	3–5 gr. 0.2–0.32 gm.
Camphor Monobromate	alm. ins.	6.5	2–5 gr. 0.13–0.32 gm.
Camphor, spts. of	....	....	10–30 m. 0.6–1.8 mil.
Capsicum, tincture	....	....	5–10 m. 0.3–0.6 mil.
Cardamom Compound Tincture of	....	....	1–2 dr. 3.75–7.5 mil.
Cardamom, tincture of	....	....	30–60 m. 1.8–3.75 mil.
Cascara Sagrada	ins.	ins.	10–30 gr. 0.6–2.0 gm.
Cascara Sagrada ext	ins.	ins.	2–8 gr. 0.13–0.5 gm.
Cascara Sagrada (fld. ext. aromatica)	....	....	15–60 m. 0.9–3.75 mil.
Castor Oil	....	....	½–2 oz. 15–60 mil.
Cerium oxalate	ins.	ins.	1–10 gr. 0.065–0.65 gm.
Chalk Mixture	....	....	4 dr. 15 mil.
Chalk Powder Compound	ins.	ins.	½–2 dr. 2.0–7.7 gm.
Chalk, prepared (Creta preparata)	ins.	ins.	10–40 gr. 0.65–2.6 gm.
Champagne (See alcohol)			
*Chenopodium oil	....	....	0.5 c.c. three times 1.5 c.c. divided into 3 doses
Chloral Hydrate	v. s.	v. s.	10–30 gr. 0.6 m.–2.0 gm.
Chloroform, spts. of	....	....	10–60 m. 0.6–3.75 mil.
Chloroform, water of	....	....	½–2 oz. 15–60 mil.
Cinchona	ins.	ins.	10–60 gr. 0.65–3.9 gm.
Cinchona, comp. tincture of	....	....	1–2 dr. 3.75–7.5 mil.
Cinnamon water	....	....	½–2 oz. 15–60 mil.
Cocain Hydrochlorid	0.4	3.2	¼–½ gr. 0.016–0.03 gm.
Codein (see Opium)			
† Cod-liver Oil (Oleum Morrhuæ)	1120	2	1–4 dr. 3.75–15 mil.

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* Recommendation of Uncinariasis Commission to the Orient. Rockefeller Foundation. "International Health Board Publication," No. 8, p. 87.

† As an antirachitic cod-liver oil should be given in from 2 to 3 times the dose in the table.

FOR INTERNAL USE (*Continued*)

Six months or 15 *lb.* 6.8 *kg.*	*One year or* 22 *lb.* 10 *kg.*	*18 months or* 25 *lb.* 11.35 *kg.*	*Two years or* 28 *lb.* 12.70 *kg.*	*Three years or* 32 *lb.* 14.64 *kg.*
15–30 m.	35–60 m.	45 m.–1½ dr.	7 m.–2 dr.	90 m.–3 dr.
1–2 mil.	2.1–4 mil.	2.7–6 mil.	4.2–8 mil.	6–12 mil.
1/26–⅕ gr.	1/13–⅓ gr.	⅛–½ gr.	⅐–¾ gr.	⅕–1 gr.
0.0025–0.013 gm.	0.004–0.02 gm.	0.008–0.03 gm.	0.009–0.048 gm.	0.013–0.065 gm.
1/10–½ gr.	⅕–⅔ gr.	⅓–1 gr.	½–1¼ gr.	¾–1½ gr.
0.006–0.02 gm.	0.013–0.04 gm.	0.02–0.065 gm.	0.03–0.08 gm.	0.048–0.09 gm.
½–¾ m.	¾–1½ m.	1–2 m.	1½–3 m.	2–4 m.
0.03–0.048 mil.	0.048–0.09 mil.	0.065–0.12 mil.	0.09–0.18 mil.	0.02–0.26 mil.
1/10–½ gr.	⅕–¾ gr.	⅓–1 gr.	½–1½ gr.	¾–2 gr.
0.006–0.06 gm.	0.013–0.048 gm.	0.02–0.065 gm.	0.03–0.065 gm.	0.048–0.12 gm.
½–¾ m.	¾–1½ m.	1–2 m.	1½–3 m.	2–4 m.
0.03–0.048 mil.	0.048–0.09 mil.	0.065–0.12 mil.	0.09–0.18 mil.	0.2–0.26 mil.
1/100–½ gr.	⅕–¾ gr.	⅓–1 gr.	½–1½ gr.	¾–2 gr.
0.006–0.03 gm.	0.013–0.048 gm.	0.02–0.065 gm.	0.03–0.065 gm.	0.048–0.12 gm.
1–2 gr.	2–4 gr.	3–6 gr.	4–8 gr.	6–12 gr.
0.065–0.12 gm.	0.12–0.26 gm.	0.18–0.36 gm.	0.26–0.48 gm.	0.36–0.73 gm.
1–2 gr.	2–4 gr.	3–6 gr.	4–8 gr.	6–12 gr.
0.065–0.12 gm.	0.12–0.26 gm.	0.18–0.36 gm.	0.26–0.48 gm.	0.36–0.72 gm.
1/100–1/26 gr.	1/50–1/13 gr.	1/30–⅛ gr.	1/25–⅐ gr.	1/20–⅕ gr.
0.0006–0.0025 gm.	0.0012–0.005 gm.	0.002–0.008 gm.	0.0024–0.009 gm.	0.003–0.013 gm.
1/150 gr.	1/75 gr.	1/50 gr.	1/40 gr.	1/30 gr.
0.0004 gm.	0.0008 gm.	0.0012 gm.	0.0015 gm.	0.002 gm.
1/12–⅕ gr.	⅙–⅓ gr.	¼–½ gr.	⅓–¾ gr.	½–1 gr.
0.005–0.013 gm.	0.01–0.02 gm.	0.016–0.03 gm.	0.02–0.048 gm.	0.03–0.065 gm.
1/12–⅕ gr.	⅙–⅓ gr.	¼–½ gr.	⅓–¾ gr.	½–1 gr.
0.005–0.013 gm.	0.01–0.02 gm.	0.016–0.03 gm.	0.02–0.048 gm.	0.03–0.065 gm.
1/10–⅕ gr.	⅕–⅓ gr.	⅓–½ gr.	½–¾ gr.	¾–1 gr.
0.006–0.013 gm.	0.013–0.02 gm.	0.02–0.03 gm.	0.03–0.048 gm.	0.048–0.065 gm.
1/12–⅕ gr.	⅙–⅓ gr.	¼–½ gr.	⅓–¾ gr.	½–1 gr.
0.005–0.013 gm.	0.01–0.02 gm.	0.016–0.03 gm.	0.02–0.048 gm.	0.03–0.065 gm.
½–1⅙ m.	¾–2½ m.	1–3½ m.	1½–4½ m.	2–6 m.
0.03–0.065 mil.	0.048–0.15 mil.	0.065–0.21 mil.	0.09–0.29 mil.	0.12–0.36 mil.
⅕–½ m.	⅓–¾ m.	½–1 m.	¾–1½ m.	1–2 m.
0.013–0.03 mil.	0.02–0.048 mil.	0.03–0.065 mil.	0.048–0.09 mil.	0.065–0.12 mil.
2–4 m.	4–8 m.	6–12 m.	8–16 m.	12–24 m.
0.12–0.26 mil.	0.26–0.48 mil.	0.36–0.72 mil.	0.48–1 mil.	0.72–1.45 mil.
1–2 m.	2–4 m.	3–6 m.	4–8 m.	6–12 m.
0.06–0.12 mil.	0.12–0.26 mil.	0.18–0.36 mil.	0.26–0.48 mil.	0.36–0.72 mil.
⅖–1 gr.	¾–2 gr.	1–3 gr.	1½–4 gr.	2–6 gr.
0.026–0.065 gm.	0.048–0.12 gm.	0.065–0.18 gm.	0.09–0.26 gm.	0.12–0.36 gm.
1/12–⅓ gr.	⅙–⅔ gr.	¼–1 gr.	⅓–1¼ gr.	½–1½ gr.
0.005–0.02 gm.	0.01–0.04 gm.	0.016–0.065 gm.	0.02–0.8 gm.	0.03–0.09 gm.
½–2 m.	1–4 m.	1½–6 m.	2–8 m.	3–12 m.
0.03–0.12 mil.	0.065–0.26 mil.	0.09–0.36 mil.	0.12–0.48 mil.	0.18–0.72 mil.
7½–30 m.	15–60 m.	25 m.–1½ dr.	35 m.–2 dr.	80 m.–3 dr.
0.45–2 mil.	1.0–4.0 mil.	1.5–6.0 mil.	2.1–8 mil.	4.8–12.0 mil.
1/26–½ gr.	1/13–¾ gr.	⅛–1 gr.	⅐–1½ gr.	⅕–2 gr.
0.025–0.03 gm.	0.005–0.048 gm.	0.008–0.065 gm.	0.009–0.09 gm.	0.013–0.12 gm.
10 m.	20 m.	30 m.	40 m.	50 m.
0.65 mil.	1.3 mil.	2.0 mil.	2.4 mil.	3.0 mil.
1–5 gr.	2½–10 gr.	3½–15 gr.	4½–20 gr.	6–25 gr.
0.065–0.3 gm.	0.15–0.65 gm.	0.21–1.0 gm.	0.29–1.3 gm.	0.36–1.5 gm.
½–1½ gr.	¾–3 gr.	1–4 gr.	1½–5 gr.	2–8 gr.
0.03–0.09 gm.	0.048–0.18 gm.	0.065–0.26 gm.	0.09–0.3 gm.	0.12–0.48 gm.
⅖–1 gr.	¾–2¼ gr.	1–3 gr.	1½–4½ gr.	2–6 gr.
0.026–0.065 gm.	0.048–0.136 gm.	0.065–0.18 gm.	0.09–0.278 gm.	0.12–0.36 gm.
½–2 m.	¾–4 m.	1–6 m.	1½–8 m.	2–12 m.
0.03–0.12 mil.	0.048–0.26 mil.	0.065–0.36 mil.	0.09–0.48 mil.	0.12–0.72 mil.
7½–30 m.	15–60 m.	25 m.–1½ dr.	35 m.–2 dr.	80 m.–3 dr.
0.45–2 mil.	1.0–4.0 mil.	1.5–6.0 mil.	2.1–8 mil.	4.8–12.0 mil.
½–2 gr.	¾–4 gr.	1–6 gr.	1½–8 gr.	2–12 gr.
0.03–0.12 gm.	0.048–0.26 gm.	0.065–0.36 gm.	0.09–0.48 gm.	0.12–0.72 gm.
2½–5 m.	5–10 m.	7½–15 m.	9–20 m.	12–25 m.
0.15–0.3 mil.	0.3–0.65 mil.	0.45–1 mil.	0.54–1.3 mil.	0.72–1.5 mil.
7½–30 m.	15–60 m.	25 m.–1½ dr.	35 m.–2 dr.	80 m.–3 dr.
0.45–2 mil.	1.0–4.0 mil.	1.5–6.0 mil.	2.1–8 mil.	4.8–12.0 mil.
1/100–1/50 gr.	1/50–1/25 gr.	1/30–1/20 gr.	1/25–1/15 gr.	1/20–1/10 gr.
0.0005–0.0012 gm.	0.0012–0.024 gm.	0.002–0.003 gm.	0.0024–0.004 gm.	0.003–0.006 gm.
2½–10 m.	5–20 m.	7–30 m.	9–40 m.	12–50 m.
0.15–0.65 mil.	0.3–1.3 mil.	0.42–2 mil.	0.54–2.4 mil.	0.72–3.0 mil.

TABLE OF DRUGS AND DOSAGE

Name	Solubility of one part		Adult dose
	In water At 77°F. At 25°C.	*In alcohol At 77°F. At 25°C.*	
Cod-liver Oil, Emulsion	....	....	2–4 dr. 7.5–15 mil.
Cod-liver Oil Phosphorized (1/25 gr. to 1 oz. cod-liver oil Phosphorus)	....	....	1–3 dr. 3.75–11.25 mil.
Corrosive sublimate	13.5	3.8	1/40–⅛ gr. 0.0015–0.008 gm.
Cream of Tartar (see Acid Tartaric) (Potassium bitartrate)			
Creosotal (Creosote carbonate)	ins.	v. s.	5–30 m. 0.3–1.8 mil.
Creosote	sl.	ins.	5–10 m. 0.3–0.6 mil.
Digalin	....	....	7–30 m. 0.5–1.8 mil.
Digipuratum	ins.	ins.	1–2 gr. 0.065–0.13 gm.
Digitalin (one-half this dose by needle)	sol.	ins.	1/10–½ gr. 0.006–0.03 gm.
Digitalis, infusion (see also External Drugs)	....	....	1–4 dr. 3.75–15 mil.
Digitalis, tincture	....	....	5–30 m. 0.3–1.8 mil.
Digitalone	....	....	10–30 m. 0.6–1.8 mil.
Dover's powder (Pulv. ipecac et opii.)	ins.	ins.	3–10 gr. 0.2–0.65 gm.
Emulsion of Asafœtida (Milk of asafœtida)	....	....	½–1 oz. 15–30 mil.
Emetin Hydrochlorid (by hypo)	v. s.	v. s.	¼–1 gr. 0.015–0.06 gm.
Epinephrin (see Adrenalin)	s.	alm. ins.	
Epsom salt	1	alm. ins.	1–8 dr. 3.9–31 gm.
Ergot, fluid extract of	....	....	½–2 dr. 1.8–8.0 mil.
Eriodictyon Aromatic syrup (Aro. Syr. yerba santa)	....	....	2 dr. 8 mil.
Ether, compound spts. of (Hoffman's anodyne)	....	....	1 dr. 4 mil.
Ether, spts. nitrous (sweet spirits of niter)	....	....	½–1 dr. 1.8–3.75 mil.
Fels Bovis Ext. (ox-gall)	s.	s.	2–8 gr. 0.12–0.5 gm.
Fennel Water	....	....	4 dr.–1 oz. 15–30 mil.
Ferric and Ferrous Preparations (see Iron):			
Fowler's solution (Liq. Potassii Arsenitis)	....	....	3–5 m. 0.2–0.3 mil.
Gallic Acid (see Acid Gallic)	87	4.6	
Gambir, Compound Tincture of	....	....	2–4 dr. 7.5–15.0 mil.
Gentian, Ext. of	ins.	ins.	2–8 gr. 0.12–0.5 gm.
Ginger, Tinct. of	....	....	8–40 m. 0.5–2.5 mil.
Glauber's salt (Sodium sulphate)	1	ins.	2 dr.–1 oz. 7.7–31 mil.
Glonoin (Nitroglycerin)	v. s.	s.	1/100 gr. 0.0006 gm.
Glonoin, spts. of	....	....	1–2 m. 0.06–0.12 mil.
Glycerin	....	....	60 m. 3.75 mil.
Glycyrrhiza (see Licorice)	....	....	30 gr. 2 gm.
Glycyrrhiza Compound Mixture (see Brown Mixture):			
Granatum (Pomegranate)	ins.	ins.	20–30 gr. 1.3–2.0 gm.
Guaiacol Carbonate	ins.	60	10–20 gr. 0.65–1.3 gm.
Heroin	1700	31	1/16–⅙ gr. 0.004–0.01 gm.
Hexamethylenamin	1.5	12.5	5–20 gr. 0.32–1.3 gm.
Hoffman's Anodyne (see compound spts. of ether)	....	....	
Hydrargyri (see Mercury):			
Hydrochloric acid dil. (see Acid, hydrochloric dil.):			
Hypophysis sicca Desiccated Pituitary Body	ins.	ins.	½–3 gr. 0.032–0.2 gm.

FOR INTERNAL USE (*Continued*)

Six months or 15 *lb.* 6.8 *kg.*	*One year or* 22 *lb.* 10.8 *kg.*	18 *months or* 25 *lb.* 11.35 *kg.*	*Two years or* 28 *lb.* 12.70 *kg.*	*Three years or* 32 *lb.* 14.64 *kg.*
5–10 m.	10–20 m.	15–30 m.	20–40 m.	25–50 m.
0.3–0.65 mil.	0.65–1.3 mil.	1.0–2 mil.	1.3–2.4 mil.	1.5–3.0 mil.
2½–7 m.	5–14 m.	7–21 m.	9–25 m.	12–36 m.
0.15–0.42 mil.	0.3–0.84 mil.	0.42–1.36 mil.	0.54–1.5 mil.	0.72–2.16 mil
1/1000–1/200 gr.	1/500–1/100 gr.	1/300–1/70 gr.	1/250–1/50 gr.	1/200–1/40 gr.
0.00006–0.0003 gm.	0.00012–0.006 gm.	0.002–0.00084 gm.	0.00024–0.0012 gm.	0.0003–0.0015 gm.
⅕–1m.	⅓–2¼ m.	½–3½ m.	¾–4¼ m.	1–6 m.
0.013–0.065 mil.	0.02–0.136 mil.	0.03–0.21 mil.	0.048–0.276 mil.	0.065–0.36 mil.
⅕–⅖ m.	⅓–¾ m.	½–1 m.	¾–1½ m.	1–2 m.
0.013–0.026 mil.	0.02–0.048 mil.	0.03–0.065 mil.	0.048–0.09 mil.	0.065–0.02 mil.
¼–1 m.	½–2¼ m.	¾–3¼ m.	1–4¼ m.	1½–6 m.
0.016–0.065 mil.	0.03–0.136 mil.	0.048–0.21 mil.	0.065–0.276 mil.	0.09–0.36 mil.
1/26–1/12 gr.	1/13–⅙ gr.	⅛–¼ gr.	1/7–⅓ gr.	⅕–½ gr.
0.0025–0.005 gm.	0.005–0.01 gm.	0.008–0.016 gm.	0.009–0.02 gm.	0.013–0.03 gm.
1/250–1/50 gr.	1/125–1/25 gr.	1/100–1/20 gr.	1/75–1/15 gr.	1/50–1/10 gr.
0.00024–0.0012 gm.	0.00048–0.0024 gm.	0.0006–0.003 gm.	0.0008–0.004 gm.	0.0012–0.006 gm.
2½–10 m.	5–20 m.	7–30 m.	9–40 m.	12–50 m.
0.15–0.65 mil.	0.3–1.3 mil.	0.42–2 mil.	0.54–2.4 mil.	0.72–3.0 mil.
⅕–1 m.	⅓–2¼ m.	½–3½ m.	¾–4¼ m.	1–6 m.
0.013–0.065 mil.	0.02–0.136 mil.	0.03–0.21 mil.	0.048–0.276 mil.	0.065–0.36 mil.
⅖–1 m.	¾–2 m.	1–3 m.	1½–4½ m.	2–6 m.
0.026–0.06 mil.	0.048–0.12 mil.	0.065–0.18 mil.	0.09–0.28 mil.	0.12–0.36 mil.
1/10–½ gr.	⅕–¾ gr.	⅓–1 gr.	½–1½ gr.	⅔–2 gr.
0.006–0.03 gm.	0.013–0.048 gm.	0.02–0.065 gm.	0.03–0.09 gm.	0.04–0.12 gm.
7½–15 m.	15–35 m.	25–45 m.	35–70 m.	80–90 m.
0.45–1 mil.	1.0–2.1 mil.	1.5–2.7 mil.	2.1–4.2 mil.	4.8–5.4 mil.
1/100–1/26 gr.	1/50–1/13 gr.	1/30–⅛ gr.	1/25–1/7 gr.	1/20–⅕ gr.
0.0006–0.0025 gm.	0.0012–0.005 gm.	0.002–0.008 gm.	0.0024–0.009 gm.	0.003–0.015 gm.
2½–15 gr.	5–30 gr.	7–45 gr.	9–70 gr.	12–90 gr.
0.15–1 gm.	0.3–2 gm.	0.42–2.7 gm.	0.54–4.2 gm.	0.72–6 gm.
1½–5 m.	3–10 m.	4½–15 m.	5–20 m.	6–25 m.
0.09–0.3 mil.	0.18–0.65 mil.	0.29–1 mil.	0.3–1.3 mil.	0.36–1.5 mil.
5 m.	10 m.	15 m.	20 m.	25 m.
0.3 mil.	0.65 mil.	1.0 mil.	1.3 mil.	1.5 mil.
2½ m.	5 m.	7 m.	9 m.	12 m.
0.15 mil.	0.3 mil.	0.42 mil.	0.54 mil.	0.72 mil.
1–2½ m.	2–5 m.	3–7 m.	4–9 m.	6–12 m.
0.065–0.15 mil.	0.12–0.03 mil.	0.18–0.47 mil.	0.26–0.54 mil.	0.36–0.72 mil.
1/12–⅓ gr.	⅙–⅔ gr.	¼–1 gr.	⅓–1¼ gr.	½–1½ gr.
0.0005–0.02 gm.	0.01–0.04 gm.	0.016–0.065 gm.	0.02–0.08 gm.	0.03–0.09 gm.
10–15 m.	20–35 m.	30–45 m.	40–70 m.	50–90 m.
0.65–1 mil.	1.3–2.1 mil.	2.0–2.7 mil.	2.4–4.2 mil.	3.0–5.4 mil.
1/10–⅕ m.	⅕–⅓ m.	⅓–½ m.	½–¾ m.	¾–1 m.
0.006–0.013 mil.	0.013–0.02 mil.	0.02–0.03 mil.	0.03–0.048 mil.	0.048–0.065 mil.
4–8 m.	8–16 m.	12–24 m.	16–32 m.	24–48 m.
0.26–0.48 mil.	0.48–1 mil.	0.72–1.45 mil.	1–2 mil.	1.45–3 mil.
1/12–⅓ gr.	⅙–⅔ gr.	¼–1 gr.	⅓–1¼ gr.	½–1½ gr.
0.005–0.02 gm.	0.01–0.04 gm.	0.016–0.065 gm.	0.02–0.08 gm.	0.03–0.09 gm.
⅓–1½ m.	⅔–3 m.	1–4 m.	1¼–5 m.	1½–8 m.
0.02–0.09 mil.	0.04–0.18 mil.	0.061–0.26 mil.	0.08–0.3 mil.	0.09–0.48 mil.
5–15 m.	10–35 m.	15–45 m.	20–70 m.	25–90 m.
0.3–1 mil.	0.65–2.1 mil.	1.0–2.7 mil.	1.3–4.2 mil.	1.5–5.4 mil.
1/2500 gr.	1/1250 gr.	1/900 gr.	1/700 gr.	1/500 gr.
0.000025 gm.	0.000048 gm.	0.000066 gm.	0.000085 gm.	0.00012 gm.
1/26–1/12 gm.	1/13–⅙ m.	⅛–¼ m.	1/7–⅓ m.	⅕–½ m.
0.0025–0.005 mil.	0.005–0.01 mil.	0.008–0.016 mil.	0.009–0.02 mil.	0.013–0.03 mil.
2 m.	4 m.	6 m.	8 m.	12 m.
0.12 mil.	0.26 mil.	0.36 mil.	0.48 mil.	0.72 mil.
1 gr.	2 gr.	3 gr.	4 gr.	6 gr.
0.065 gm.	0.12 gm.	0.18 gm.	0.26	0.36 gm.
¾–1 gr.	1½–2½ gr.	2¼–3½ gr.	3–4½ gr.	4–6 gr.
0.048–0.065 gm.	0.09–0.15 gm.	0.136–0.21 gm.	0.18–0.29 gm.	0.26–0.35 gm.
½–¾ gr.	¾–1½ gr.	1–2¼ gr.	1½–3 gr.	2–4 gr.
0.03–0.048 gm.	0.048–0.09 gm.	0.065–0.136 gm.	0.09–0.18 gm.	0.12–0.26 gm.
1/400–1/160 gr.	1/200–1/75 gr.	1/140–1/50 gr.	1/100–1/40 gr.	1/80–1/30 gr.
0.00015–0.0004 gm.	0.0003–0.0008 gm.	0.00042–0.0012 gm.	0.0006–0.0015 gm.	0.00075–0.002 gm.
⅕–1 gr.	⅓–2 gr.	½–3 gr.	¾–4 gr.	1–6 gr.
0.013–0.065 gm.	0.02–0.12 gm.	0.03–0.18 gm.	0.048–0.26 gm.	0.065–0.36 gm.
1/50–1/10 gr.	1/25–⅕ gr.	1/16–⅓ gr.	1/14–½ gr.	1/10–⅔ gr.
0.0012–0.006 gm.	0.0024–0.013 gm.	0.0037–0.02 gm.	0.0035–0.03 gm.	0.001–0.04 gm.

TABLE OF DRUGS AND DOSAGE

Name	*Solubility of one part*		*Adult dose*
	In water At 77°F. At 25°C.	*In alcohol At 77°F. At 25°C.*	
Iodid Ammonium	0.6	3.7	3–10 gr. 0.2–0.65 gm.
Iodid Potassium	0.7	22	5–20 gr. 0.32–1.3 gm.
Iodid Sodium	0.55	2	5–30 grs. 0.32–2.0 gm.
Iodid, Syrup Iron (see Iron):			
Ipecac, Powder	ins.	ins.	1 gr. 0.06 gm.
Ipecac, Syr. of (as emetic)	....	....	4 dr. 15 mil.
Ipecac, Syr. of (as expectorant)	....	....	15–30 m. 0.9–1.8 mil.
Ipecac, wine of	....	....	15 m. 1 mil.
Iron Preparations:			
Ferri carbonas saccharatus (Saccharated ferrous carbonate)	ins.	ins.	5–15 gr. 0.32–1.0 gm.
Ferric chlorid	v. s.	v. s.	1 gr. 0.065 gm.
Ferric chlorid, tinct. of	....	....	5–30 m. 0.3–1.8 mil.
Ferric citrate	s.	ins.	3–10 gr. 0.2–0.65 gm.
Vallet's Mass (Mass ferrous carbonate)	s.	ins.	3–5 gr. 0.2–0.32 gm.
Ferrous iodid, syr. of	....	....	10–40 m. 0.6–2.5 mil.
Ferrous lactate	40	ins.	15 gr. 1 gm.
Iron Oxid saccharated	....	ins.	15 gr. 1 gm.
Ferric phosphate	v. s.	ins.	5–10 gr. 0.32–0.65 gm.
Liq. ferri et ammonium acetate (Basham's mixture)	....	....	2–4 dr. 7.5–15 mil.
Ferrum reductum	ins.	ins.	1–3 gr. 0.065–0.20 gm.
Ferric Pyrophosphate	v. s.	ins.	2–10 gr. 0.13–0.65 gm.
Ferri, quininæ et strychininæ Phos. elixir	....	....	1–2 dr. 3.75–7.5 mil.
Ferri sulphas exsiccatus (Dry ferrous sulphate)	1.4	ins.	1–3 gr. 0.065–0.2 gm.
Ferric Citrate (Hypo)	s.	ins.	1 gr. 0.065 gm.
Blaud's pills	....	....	5–20 gr. (2–6 pills) 0.32–1.3 gm.
Jalap Powder	ins.	ins.	10–20 gr. 0.65–1.3 gm.
Jalap Compound Powder	ins.	ins.	30–60 gr. 2.0–3.9 gm.
Jalap Compound Tincture	....	....	½–1 dr. 1.8–3.75 mil.
Kino tincture	....	....	1–2 dr. 3.75–7.5 mil.
Krameriae, tincture	....	....	1–2 dr. 3.75–7.5 mil.
Lactic Acid	....	....	20–30 m. 1.3–1.8 mil.
Laudanum (see Opium)	....	....	5–10 m. 0.3–0.6 mil.
Licorice (see Glycyrrhizae):			
Licorice Compound Mixture (Brown Mixture)	....	....	½–1 oz. 15–30 mil.
Licorice Compound Powder	ins.	ins.	30–60 gr. 2.0–3.9 gm.
Lime water (Liquor calcis)	....	....	1–4 oz. 30–120 mil.
Liquid Petrolatum (Russian oil)	....	....	½–3 oz. 15–90 mil.
Liquor Hypophysis U.S.P. (Obstetrical pituitrin, etc. Commercial solutions are two to five times stronger. Dose should consequently be from one-half to one-fifth weaker.	....	....	10–30 m. 0.6–1.8 mil.
Lithium Bromid	0.6	v. s.	10–20 gr. 0.65–1.3 gm.
Lithium Carbonate	78	alm. ins.	5–15 gr. 0.32–1.0 gm.

FOR INTERNAL USE (*Continued*)

Six months or 15 *lb.* 6.8 *kg.*	*One year or* 22 *lb.* 10.8 *kg.*	18 *months or* 25 *lb.* 11.35 *kg.*	*Two years or* 28 *lb.* 12.70 *kg.*	*Three years or* 32 *lb.* 14.64 *kg.*
⅒–½ gr.	⅕–¾ gr.	⅓–1 gr.	½–1½ gr.	¾–2 gr.
0.006–0.03 gm.	0.013–0.048 gm.	0.02–0.065 gm.	0.03–0.065 gm.	0.048–0.12 gm.
⅕–¾ gr.	⅓–1½ gr.	½–2 gr.	¾–3 gr.	1–4 gr.
0.013–0.048 gm.	0.02–0.09 gm.	0.03–0.12 gm.	0.048–0.18 gm.	0.065–0.26 gm.
⅕–1 gr.	⅓–2 gr.	½–3 gr.	¾–4 gr.	1–6 gr.
0.013–0.065 gm.	0.02–0.12 gm.	0.03–0.18 gm.	0.048–0.26 gm.	0.065–0.36 gm.
1/26 gr.	1/13 gr.	⅛ gr.	⅐ gr.	⅕ gr.
0.0025 gm.	0.005 gm.	0.008 gm.	0.009 gm.	0.013 gm.
10 m.	20 m.	30 m.	40 m.	50 m.
0.65 mil.	1 3 mil.	2.0 mil.	2.4 mil.	3.0 mil.
½–1 m.	1-2 m.	1½–3 m.	2–4 m.	3–6 m.
0.03–0.065 mil.	0.065–0.12 mil.	0.09–0.18 mil.	0.013–0.26 mil.	0.18–0.36 mil.
½ m.	1 m.	2 m.	2½ m.	3 m.
0.03 mil.	0.065 mil.	0.12 mil.	0.15 mil.	0.18 mil.
⅕–½ gr.	⅓–1 gr.	½–1½ gr.	¾–2 gr.	1–3 gr.
0.013–0.03 gm.	0.02–0.065 gm.	0.03–0.09 gm.	0.048–0.12 gm.	0.065–0.18 gm.
1/150 gr.	1/13 gr.	⅛ gr.	⅐ gr.	⅕ gr.
0.0004 gm.	0.005 gm.	0.008 gm.	0.009 gm.	0.013 gm.
⅕–1 m.	⅓–2 m.	½–3 m.	¾–4 m.	1–6 m.
0.013–0.065 mil.	0.02–0.12 mil.	0.03–0.18 mil.	0.048–0.26 mil.	0.065–0.36 mil.
⅒–1 gr.	⅕–2½ gr.	⅓–3½ gr.	½–4½ gr.	⅔–6 gr.
0.006–0.065 gm	0.013-0.15 gm.	0.02–0.21 gm.	0.03–0.29 gm.	0.04–0.36 gm.
⅒–⅕ gr.	⅕–⅓ gr.	⅓–½ gr.	½–¾ gr.	¾–1 gr.
0.006–0 013 gm.	0.013–0.02 gm.	0.03–0.02 gm.	0.03–0.048 gm.	0.048–0.065 gm.
1 m.	2½ m.	3½ m.	4½ m.	6 m.
0.065 mil.	0.15 mil.	0.21 mil.	0.29 mil.	0.36 mil.
½ gr.	1 gr.	1½ gr.	2 gr.	3 grs.
0.03 gm.	0.065 gm.	0.09 gm.	0.12 gm.	0.18 gm.
½ gr.	1 gr.	1½ gr.	2 gr.	3 gr.
0.03 gm.	0.065 gm.	0.09 gm.	0.12 gm.	0.18 gm.
⅕–1 gr.	⅓–2½ gr.	½–3½ gr.	¾–4½ gr.	1–6 gr.
0.013–0.065 gm.	0.02–0.15 gm.	0.03–0.21 gm.	0.048–0.29 gm.	0.065–0.36 gm.
5–10 m.	10–20 m.	15–30 m.	20–40 m.	25–50 m.
0.3–0.65 mil.	0.65–1.3 mil.	1.0–2.0 mil.	1.3–2.4 mil.	1.5–3.0 mil.
1/26–⅒ gr.	1/13–⅕ gr.	⅛–⅓ gr.	⅐–½ gr.	⅕–¾ gr.
0.0025–0.006 gm.	0.005–0.0013 gm.	0.008–0.02 gm.	0.0009–0.03 gm.	0.013–0.048 gm.
1/12–1 gr.	⅙–2½ gr.	¼–3½ gr.	⅓–4½ gr.	½–6 gr.
0.005–0.065 gm.	0.01–0.15 gm.	0.016–0.21 gm.	0.02–0.29 gm.	0.03–0.36 gm.
2½–5 m.	5–10 m.	7–15 m.	9–20 m.	12–25 m.
0.15–0.3 mil.	0.3–0.65 mil.	0.42–1 mil.	0.54–1.3 mil.	0.72–1.5 mil.
1/150–⅒ gr.	1/13–⅕ gr.	⅛–⅓ gr.	⅐–½ gr.	⅕–⅔ gr.
0.0004–0.006 gm.	0 005–0.013 gm.	0.008–0.02 gm.	0.009–0.03 gm.	0.013–0.04 gm.
1/150 gr.	1/13 gr.	⅛ gr.	⅐ gr.	⅕ gr.
0.0004 gm.	0.005 gm.	0.008 gm.	0.009 gm.	0.013 gm.
⅕–¾ gr.	⅓–1½ gr.	½–2 gr.	¾–3 gr.	1–4 gr.
0.013–0.048 gm.	0.02–0.09 gm.	0.03–0.12 gm.	0.048–0.18 gm.	0.065–0.26 gm.
½–¾ gr.	¾-1½ gr.	1–2 gr.	1½–3 gr.	2–4 gr.
0.03–0.048 gm.	0.048–0.09 gm.	0.065–0.12 gm.	0.09–0.18 gm.	0.12–0.26 gm.
1–2 gr.	2–4 gr.	3–6 gr.	4–8 gr.	6–12 gr.
0.065–0.12 gm.	0.12–0.26 gm.	0.18–0.36 gm.	0.24–0.48 gm.	0.36–0.72 gm.
1-2 m.	2–4 m.	3–6 m.	4–8 m.	6–12 m.
0.065-0.12 mil.	0.12–0.26 mil.	0.18–0.36 mil.	0.24–0.48 mil.	0.36–0.72 mil.
2½–5 m.	5–10 m.	7–15 m.	9–20 m.	12–25 m.
0.15–0.3 mil	0.3–0.65 mil.	0.42–1.0 mil.	0.54–1.3 mil.	0.72–1.5 mil.
2½–5 m.	5–10 m.	7–15 m.	9–20 m.	12–25 m.
0.15–0.3 mil.	0.3–0.65 mil.	0.42–1.0 mil.	0.54–1.3 mil.	0.72–1.5 mil.
¾–1 m.	1½–2 m.	2–3 m.	3–4 m.	4–6 m.
0.048–0.065 mil.	0.09–0.12 mil.	0.12–0.18 mil.	0.18–0.26 mil.	0.26–0.36 mil.
⅕–½ m.	⅓–¾ m.	½–1 m.	¾–1½ m.	1–2 m.
0.013–0.03 mil.	0.02–0.048 mil.	0.03–0.065 mil.	0.048–0.09 mil.	0.065–0.12 mil.
7½–15 m.	17–35 m.	25–45 m.	35–70 m.	80–90 m.
0.45–1 mil.	1.02–2.1 mil.	1.5–2.7 mil.	2.1–4.2 mil.	4.8–5.4 mil.
1–2 gr.	2½–4½ gr.	3½–6½ gr.	4½–8½ gr.	6–12 gr.
0.06–0.12 gm.	0.15–0.29 gm.	0.21–0.39 gm.	0.29–0.51 gm.	0.36–0.72 gm.
20 m.–1 dr.	30 m.–2 dr.	40 m.–3 dr.	1–4 dr.	1½–6 dr.
1.3–4.0 mil.	2.0–7.8 mil.	3.3–12.0 mil.	4.0–16 mil.	6.0–24 mil.
10 m.–1 dr.	20 m.–2 dr.	30 m.–3 dr.	40 m.–3½ dr.	50 m.–5 dr.
0.65–3.9 mil.	1.3–7.8 mil.	2.0–11.7 mil.	2.65–13.5 mil.	3.0–19.5 mil.
⅖–1 m.	¾–2 m.	1–3 m.	1½–4½ m.	2–6 m.
0.026–0.06 mil.	0.048–0.12 mil.	0.065–0.18 mil.	0.09–0.28 mil.	0.12–0.36 mil.
½–¾ gr.	¾–1½ gr.	1–2 gr.	1½–3 gr.	2–4 gr.
0.03–0.048 gm.	0.048–0.09 gm.	0.065–0.12 gm.	0.09–0.18 gm.	0.12–0.26 gm.
⅕–½ gr.	⅓–1 gr.	½–1½ gr.	¾–2 gr.	1–3 gr.
0.013–0.03 gm.	0.02–0.065 gm.	0.03–0.09 gm.	0.048–0.12 gm.	0.065–0.18 gm.

TABLE OF DRUGS AND DOSAGE

Name	*Solubility of one part*		*Adult dose*
	In water At 77°F. At 25°C.	*In alcohol At 77°F. At 25°C.*	
Lithium Citrate	1.4	alm. ins.	10–30 gr. 0.65–2.0 gm.
Magnesium Carbonate (as antacid)	ins.	ins.	5–20 gr. 0.32–1.3 gm.
Magnesium Carbonate (as laxative)	ins.	ins.	½–2 dr. 2–7.7 gm.
Magnesia Citrate Sol. (as laxative)	....	....	6 oz. 180 mil.
Magnesia citrate sol. (as purgative)	....	....	12 oz. 360 mil.
Magnesium oxid (as antacid)	alm. ins.	ins.	5–20 gr. 0.32–1.3 gm.
Magnesium oxid (as laxative)	alm. ins.	ins.	½–1 dr. 2–3 gm.
Magnesia, Milk of (as antacid)	....	....	1–4 dr. 3.75–15 mil.
Magnesia, Milk of (Magnesia Magma) (as purgative)	....	....	1–3 oz. 60–180 mil.
Magnesium Sulphate	1	alm. ins.	2 dr.–1 oz. 7.7–31 gm.
Male fern Oleoresin	....	....	½–1 fl. dr. 1.8–3.75 mil.
Malt, extract of	....	....	1–4 dr. 3.75–15 mil.
Manna	s.	s.	½–1 oz. 15.5–31 gm.
Mentha Piperita water	As vehicle of drugs		Q. S.
Mentha Virides water	As vehicle of drugs		Q. S.
Mercury:			
Mass of Mercury (as alterative)	ins.	ins.	1–3 gr. 0.065–0.2 gm.
Mercury Corrosive Chlorid	13.5	3.8	1/40–1/8 gr. 0.0015–0.008 gm.
Mercury, Mild Chlorid (see Calomel):			
Mercury, Red Iodid (Biniodid of Mercury)	alm. ins.	115	1/20–1/12 gr. 0.003–0.005 gm.
Mercury with chalk (as alterative)	ins.	ins.	1–3 gr. 0.06–0.2 gm.
Mercury with chalk (Hydrargyrum Cum Creta) (as laxative)	ins.	ins.	10–20 gr. 0.65–1.3 gm.
Methyl Salicylate	sp. s.	v. s.	5–20 m. 0.3–1.2 mil.
Mindererus, spts. of (Liq. Amm. Acet.)	....	....	½–1 oz. 15–30 mil.
Morphin Sulphate	15.5	565	1/12–½ gr. 0.005–0.032 gm.
Myrrh, Tinct.	....	....	15–30 m. 0.9–1.8 mil.
Mustard	ins.	ins.	1–2 dr. diluted in water is prompt emetic 30–60 gm.
Niter, Sweet spts. of	....	....	½–1 dr. 1.8–3.75 mil.
Nitroglycerin	v. sl. s.	s.	1/100 gr. 0.0006 gm.
Spts. of Glyceryl Trinitrate (Spts. glonoin)	....	....	1–2 m. 0.06–0.12 mil.
Novaspirin	ins.	s.	10–30 gr. 0.62–2 gm.
Nux Vomica	ins.	ins.	1–4 gr. 0.065–0.26 gm.
Strychnin	6420	136	1/60–1/15 gr. 0.0011–0.004 gm.
Nux Vomica, Tinct, of	....	....	10–30 m. 0.6–1.8 mil.
Oleum Gaultheriae (Oil of Wintergreen)	....	....	10–20 m. 0.6–1.3 mil.
Oleum Morrhaue (see Cod-liver Oil)	....	....	1–4 dr. 3.75–15 mil.
Oleum Olivæ	....	....	1–2 oz. 30–60 mil.
Oleum Ricini	....	....	½–2 oz. 15–60 mil.
Opium:			
Opium, Tincture Camphorated (Paregoric)	....	....	1–4 dr. 3.75–15.0 mil.
Opium, Tincture Deodorized	....	....	5–10 m. 0.3–0.6 mil.

FOR INTERNAL USE (*Continued*)

Six months or 15 *lb.* 6.8 *kg.*	*One year or* 22 *lb.* 10.8 *kg.*	18 *months or* 25 *lb.* 11.35 *kg.*	*Two years or* 28 *lb.* 12.70 *kg.*	*Three years or* 32 *lb.* 14.64 *kg.*
½–1 gr.	¾–2½ gr.	1–3½ gr.	1½–4½ gr.	2–6 gr.
0.03–0.065 gm.	0.048–0.15 gm.	0.065–0.21 gm.	0.09–0.29 gm.	0.12–0.36 gm.
⅕–¾ gr.	⅓–1½ gr.	½–2 gr.	¾–3 gr.	1–4 gr.
0.012–0.048 gm.	0.021–0.09 gm.	0.03–0.12 gm.	0.048–0.18 gm.	0.065–0.26 gm.
1–5 gr.	2½–10 gr.	3½–15 gr.	4½–20 gr.	6–25 gr.
0.065–0.3 gm.	0.15–0.65 gm.	0.21–1.0 gm.	0.29–1.3 gm.	0.36–1.5 gm.
¼ oz.	½ oz.	¾ oz.	1 oz.	1¼ oz.
8.0 mil.	16.0 mil.	24.0 mil.	30 mil.	38 mil.
½ oz.	1 oz.	1½ oz.	1¾ oz.	2 oz.
16.0 mil.	30 mil.	46 mil.	54 mil.	60 mil.
⅕–¾ gr.	⅓–1½ gr.	½–2 gr.	¾–3 gr.	1–4 gr.
0.012–0.048 gm.	0.021–0.09 gm.	0.03–0.12 gm.	0.048–0.18 gm.	0.065–0.26 gm.
1–2 gr.	2½–4½ gr.	3½–6½ gr.	4½–8½ gr.	6–12 gr.
0.065–0.12 gm.	0.15–0.29 gm.	0.21–0.39 gm.	0.29–0.51 gm.	0.36–0.72 gm.
2–10 m.	4–20 m.	6–30 m.	8–40 gm.	12–50 gm.
0.12–0.65 mil.	0.26–1.3 mil.	0.36–2.0 mil.	0.48–2.4 mil.	0.72–3.0 mil.
20–60 m.	40–120 m.	60–180 m.	80–240 m.	100–300 m.
1.3–3.75 mil.	2.6–7.7 mil.	3.75–11.25 mil.	5–15 mil.	6.3–19 mil.
5–15 m.	10–35 m.	15–45 m.	20–70 m.	25–90 m.
0.3–1 mil.	0.65–2.1 mil.	1.0–2.7 mil.	1.03–4.2 mil.	1.5–6.0 mil.
1½–2½ m.	3–5 m.	4½–7½ m.	5–9 m.	6–12 m.
0.09–0.15 mil.	0.18–0.3 mil.	0.29–0.45 mil.	0.3–0.54 mil.	0.36–0.72 mil.
2–8 m.	4–16 m.	6–24 m.	8–32 m.	12–48 m.
0.12–0.48 mil.	0.26–1 mil.	0.36–1.45 mil.	0.48–2 mil.	0.72–3 mil.
7½–15 gr.	17–30 gr.	25–45 gr.	35–70 gr.	40–90 gr.
0.45–1 gm.	1.02–2 gm.	1.5–2.7 gm.	2.1–4.2 gm.	2.4–6 gm.
1/26–⅛ gr.	1/13–¼ gr.	⅛–¾ gr.	1/7–½ gr.	⅕–⅔ gr.
0.0025–0.008 gm.	0.005–0.016 gm.	0.008–0.048 gm.	0.009–0.03 gm.	0.013–0.04 gm.
1/1000–1/200 gr.	1/500–1/100 gr.	1/300–1/60 gr.	1/250–1/50 gr.	1/200–1/40 gr.
0.00006–0.0003 gm.	0.00012–0.0006 gm.	0.0002–0.001 gm.	0.00024–0.0012 gm.	0.0003–0.00015 gm.
1/500–1/300 gr.	1/250–1/150 gr.	1/200–1/100 gr.	1/150–1/80 gr.	1/100–1/60 gr.
0.00012–0.0002 gm.	0.00024–0.0004 gm.	0.0003–0.0006 gm.	0.0004–0.00075 gm.	0.0006–0.001 gm.
1/26–⅛ gr.	1/13–¼ gr.	⅕–¾ gr.	1/7–½ gr.	⅕–⅔ gr.
0.0025–0.008 gm.	0.005–0.016 gm.	0.008–0.048 gm.	0.009–0.03 gm.	0.013–0.04 gm.
½–¾ gr.	¾–1½ gr.	1–2 gr.	1½–3 gr.	2–4 gr.
0.03–0.048 gm.	0.048–0.09 gm.	0.065–0.12 gm.	0.09–0.18 gm.	0.12–0.26 gm.
⅕–¾ m.	⅓–1½ m.	½–2 m.	¾–3 m.	1–4 m.
0.013–0.048 mil.	0.02–0.09 mil.	0.03–0.12 mil.	0.048–0.18 mil.	0.065–0.26 mil.
7½–15 m.	17–30 m.	25–45 m.	35–70 m.	40–90 m.
0.45–1.0 mil.	1.02–2.0 mil.	1.5–2.7 mil.	2.1–4.2 mil.	2.4–6 mil.
1/300–1/50 gr.	1/150–1/25 gr.	1/100–1/16 gr.	1/80–1/15 gr.	1/60–1/10 gr.
0.0002–0.0012 gm.	0.0004–0.0024 gm.	0.0006–0.0037 gm.	0.00075–0.004 gm.	0.001–0.006 gm.
½–1 m.	1–2 m.	1½–3 m.	2–4 m.	3–6 m.
0.03–0.065 mil.	0.065–0.12 mil.	0.09–0.18 mil.	0.13–0.26 mil.	0.18–0.36 mil.
2½–5 gr.	5–10 gr.	7–15 gr.	9–18 gr.	12–25 gr.
0.15–0.3 gm.	0.3–0.65 gm.	0.42–1 gm.	0.54–1.08 gm.	0.72–1.5 gm.
1½–2½ m.	3–5 m.	4½–7½ m.	5–9 m.	6–12 m.
0.09–0.15 mil.	0.18–0.3 mil.	0.29–0.45 mil.	0.3–0.54 mil.	0.36–0.72 mil.
1/2500 gr.	1/1250 gr.	1/900 gr.	1/700 gr.	1/500 gr.
0.000025 gm.	0.000048 gm.	0.000066 gm.	0.000085 gm.	0.00012 gm.
1/26–1/12 m.	1/13–⅙ m.	⅛–¼ m.	1/7–⅓ m.	⅕–½ m.
0.0025–0.005 mil.	0.005–0.01 mil.	0.008–0.016 mil.	0.009–0.02 mil.	0.013–0.03 mil.
⅖–1 gr.	¾–2 gr.	1–3 gr.	1½–4 gr.	2–6 gr.
0.026–0.065 gm.	0.048–0.12 gm.	0.065–0.18 gm.	0.09–0.26 gm.	0.12–0.36 gm.
1/26–⅙ gr.	1/13–⅓ gr.	⅛–½ gr.	1/7–¾ gr.	⅕–1 gr.
0.0025–0.01 gm.	0.005–0.02 gm.	0.008–0.03 gm.	0.009–0.048 gm.	0.013–0.065 gm.
1/1600–1/400 gr.	1/800–1/200 gr.	1/500–1/150 gr.	1/400–1/100 gr.	1/300–1/75 gr.
0.000037–0.00015 gm.	0.000075–0.0003 gm.	0.00012–0.0004 gm.	0.00015–0.0006 gm.	0.0002–0.0008 gm.
½–1 m.	¾–2 m.	1–3 m.	1½–4 m.	2–6 m.
0.03–0.06 mil.	0.048–0.12 mil.	0.065–0.18 mil.	0.09–0.26 mil.	0.12–0.36 mil.
½–¾ m.	¾–1½ m.	1–2 m.	1½–3 m.	2–4 m.
0.03–0.048 mil.	0.048–0.09 mil.	0.065–0.12 mil.	0.09–0.18 mil.	0.12–0.26 mil.
2–10 m.	5–20 m.	7–30 m.	9–40 m.	12–50 m.
0.12–0.65 mil.	0.3–1.3 mil.	0.42–2.0 mil.	0.54–2.4 mil.	0.72–3.0 mil.
15–30 m.	35–60 m.	45 m.–1½ dr.	70 m.–2 dr.	90 m.–3 dr.
1–2 mil.	2.1–4 mil.	2.7–6 mil.	4.2–8 mil.	6–12 mil.
7½–30 m.	15–60 m.	25 m.–1½ dr.	35 m.–2 dr.	80 m.–3 dr.
0.45–2 mil.	1.0–4.0 mil.	1.5–6.0 mil.	2.1–8 mil.	4.8–12.0 mil.
2½–10 m.	5–20 m.	7–30 m.	9–40 m.	12–50 m.
0.15–0.65 mil.	0.3–1.3 mil.	0.42–2.0 mil.	0.54–2.4 mil.	0.72–3.0 mil.
⅕–⅖ m.	⅓–¾ m.	½–1 m.	¾–1½ m.	1–2 m.
0.013–0.026 mil.	0.02–0.048 mil.	0.03–0.065 mil.	0.048–0.09 mil.	0.065–0.12 mil.

TABLE OF DRUGS AND DOSAGE

Name	*Solubility of one part*		*Adult dose*
	In water At 77°F. At 25°C.	*In alcohol At 77°F. At 25°C.*	
Opium Tincture (Laudanum)	....	....	5–10 m. 0.3–0.6 mil.
Powdered ipecac and opium	ins.	ins.	3–10 gr. 0.2–0.65 gm.
Codein	120	2	¼–2 gr. 0.015–0.13 gm.
Heroin	1700	31	1⁄12–1⁄6 gr. 0.005–0.01 gm.
Morphin Sulphate	3340	210	1⁄12–½ gr. 0.005–0.032 gm.
Orange Juice	....	....	6–12 oz. 22.5–45 mil.
Ox-gall (see Fels Bovis)	s.	s.	2–8 gr. 0.12–0.5 gm.
Pancreatin	ins.	s.	8–15 gr. 0.5–1.0 gm.
Paregoric (Tincture opium camphorated)	....	....	1–4 dr. 3.75–15.0 mil.
Pepo (Pumpkin seed)	ins.	ins.	1–2 oz. 31–62 gm.
Pelletierin Tannate	240	1.6	3–5 gr. 0.2–0.3 gm.
Pelletierin (Tanret's)	....	....	1 oz. 30 mil.
Pepsin, Powder	50	alm. ins.	10–60 gr. 0.65–3.9 gm.
Pepsin, Essence of	....	....	1–2 dr. 4–8 mil.
Petroleum Liquid (see Liquid Petrolatum):			
Phenacetin	1310	15	5–15 gr. 0.32–1.0 gm.
Phenolphthalein	13	alm. ins.	½–3 gr. 0.032–0.2 gm.
Phosphorus:			
Phosphoratum Oleum (Phosphorated Oil)	....	....	1–2 m. 0.06–0.13 mil.
Phosphoric acid dil. (see Acid, phosphoric dil.):			
Hypophosphite Comp. Syr. of	....	....	1–2 dr. 4–8 mil.
Lactophosphate, syrup calcium	....	....	1–2 dr. 4–8 mil.
Philocarpin (not advised in the treatment of children):			
Piperita, Aqua Mentha	Q. S.		
Podophyllum	ins.	ins.	3–5 gr. 0.2–0.32 gm.
Potassium Acetate	0.5	2.9	15–60 gr. 1–3.9 gm.
Potassium Bicarbonate	2.8	alm. ins.	10–60 gr. 0.65–3.9 gm.
Potassium Bitartrate (see Acid, tartaric):			
Potassium Bromid	1.5	250	10–45 gr. 0.65–3 gm.
Potassium Chlorate	0.6	alm. ins.	5–15 gr. 0.32–1 gm.
Potassium Citrate	11.5	alm. ins.	20–60 gr. 1.3–3.9 gm.
Potassium Iodid (anti syphlitic)	0.7	22	5–30 gr. 0.32–2 gm.
Potassium Iodid (expectorant)	0.7	22	5–20 gr. 0.32–1.3 gm.
Potassium and Sodium Tartrate (see Acid, tartaric):			
Prunus Virginiana Syrup of (Syrup of Wild Cherry)	....	....	1–4 dr. 3.75–15 mil.
Pumpkin Seed (see Pepo):			
Quassia Infusion (see External Drugs):			
Quinin Bisulphate (antimalarial)	9	23	2–15 gr. 0.12–1 gm.
Quinin Bisulphate (tonic)	9	23	1–2 gr. 0.065–0.12 gm.
Quinin sulphate (tonic)	725	107	1–2 gr. 0.065–0.12 gm.
Quinin Sulphate (antimalarial)	725	107	1–10 gr. 0.065–0.65 gm.
Cinchona, Tincture	....	....	1–2 dr. 3.75–7.5 mil.
Rhubarb:			
Rhubarb and Magnesia Anisated Powder	ins.	ins.	30–60 gr. 2–4 gm.

FOR INTERNAL USE (*Continued*)

Six months or 15 *lb.* 6.8 *kg.*	*One year or* 22 *lb.* 10.8 *kg.*	18 *months or* 25 *lb.* 11.35 *kg.*	*Two years or* 28 *lb.* 12.70 *kg.*	*Three years or* 32 *lb.* 14.64 *kg.*
⅕–½ m.	⅓–¾ m.	½–1 m.	¾–1½ m.	1–2 m.
0.013–0.03 mil.	0.02–0.048 mil.	0.03–0.065 mil.	0.048–0.09 mil.	0.065–0.12 mil.
⅒–½ gr.	⅕–¾ gr.	⅓–1 gr.	½–1½ gr.	⅔–2 gr.
0.006–0.03 gm.	0.013–0.048 gm.	0.02–0.065 gm.	0.03–0.09 gm.	0.04–0.12 gm.
1/100–1/12 gr.	1/50–⅙ gr.	1/30–¼ gr.	1/25–⅓ gr.	1/20–½ gr.
0.0006–0.005 gm.	0.0012–0.01 gm.	0.002–0.016 gm.	0.0024–0.02 gm.	0.003–0.03 gm.
1/300–1/150 gr.	1/150–1/75 gr.	1/100–1/50 gr.	1/80–1/40 gr.	1/60–1/30 gr.
0.0002–0.0004 gm.	0.0004–0.0008 gm.	0.0006–0.0012 gm.	0.00075–0.0015 gm.	0.001–0.002 gm.
1/300–1/50 gr.	1/150–1/25 gr.	1/100–1/16 gr.	1/80–1/14 gr.	1/60–⅒ gr.
0.0002–0.0012 gm.	0.0004–0.0024 gm.	0.0006–0.0037 gm.	0.00075–0.0035 gm.	0.006–0.001 gm.
2–4 dr.	4–8 dr.	6–12 dr.	8–16 dr.	10–20 dr.
7.7–15 mil.	15–30 mil.	22.5–45 mil.	30–60 mil.	37.5–75 mil.
1/12–⅓ gr.	⅙–⅔ gr.	¼–1 gr.	⅓–1¼ gr.	½–1½ gr.
0.005–0.02 gm.	0.01–0.04 gm.	0.016–0.065 gm.	0.02–0.08 gm.	0.03–0.09 gm.
⅓–½ gr.	⅔–1 gr.	1–2 gr.	1¼–2½ gr.	1½–3 gr.
0.02–0.03 gm.	0.04–0.065 gm.	0.065–0.12 gm.	0.08–0.15 gm.	0.09–0.18 gm.
2½–10 m.	5–20 m.	7–30 m.	9–40 m.	12–50 m.
0.15–0.65 mil.	0.3–1.3 mil.	0.42–2 mil.	0.54–2.4 mil.	0.72–3.0 mil.
15–30 gr.	35–60 gr.	45 gr.–1½ dr.	70 gr.–2 dr.	1½–3 dr.
1.0–2 gm.	2.1–4 gm.	2.7–6 gm.	4.2–8 gm.	6–12 gm.
⅛–⅕ gr.	¼–⅓ gr.	⅓–½ gr.	⅖–¾ gr.	½–1 gr.
0.008–0.013 gm.	0.016–0.02 gm.	0.02–0.03 gm.	0.026–0.048 gm.	0.03–0.065 gm.
15 m.	35 m.	45 m.	70 m.	90 m.
1 mil.	2.1 mil.	2.7 mil.	4.2 mil.	5.4 mil.
⅖–2 gr.	¾–4 gr.	1–6 gr.	1½–8 gr.	2–12 gr.
0.026–0.12 gm.	0.048–0.26 gm.	0.065–0.36 gm.	0.09–0.48 gm.	0.12–0.72 gm.
2½–4 m.	5–10 m.	7–15 m.	9–20 m.	12–25 m.
0.15–0.3 mil.	0.3–0.65 mil.	0.42–1 mil.	0.54–1.3 mil.	0.72–1.5 mil.
⅕–½ gr.	⅓–1 gr.	½–2 gr.	¾–2 gr.	1–3 gr.
0.013–0.03 gm.	0.02–0.065 gm.	0.03–0.12 gm.	0.048–0.12 gm.	0.065–0.18 gm.
1/50–⅛ gr.	1/25–¼ gr.	1/16–⅓ gr.	1/14–⅖ gr.	⅒–½ gr.
0.0012–0.008 gm.	0.0024–0.016 gm.	0.0037–0.02 gm.	0.004–0.026 gm.	0.006–0.03 gm.
1/26–1/12 m.	1/13–⅙ m.	⅛–¼ m.	⅐–⅓ m.	⅕–½ m.
0.0025–0.005 mil.	0.005–0.01 mil.	0.008–0.0016 mil.	0.009–0.02 mil.	0.013–0.03 mil.
2–5 m.	4½–10 m.	6½–15 m.	8½–20 m.	12–25 m.
0.12–0.3 mil.	0.29–0.65 mil.	0.39–1.0 mil.	0.51–1.3 mil.	0.72–1.5 mil.
2½–5 m.	5–10 m.	7½–15 m.	9–18 m.	12–25 m.
0.15–0.3 mil.	0.3–0.6 mil.	0.45–1 mil.	0.54–1.08 mil.	0.72–1.5 mil.
⅒–⅕ gr.	⅕–⅓ gr.	⅓–½ gr.	½–¾ gr.	¾–1 gr.
0.006–0.012 gm.	0.013–0.021 gm.	0.02–0.03 gm.	0.03–0.048 gm.	0.048–0.065 gm.
½–2 gr.	1–4½ gr.	2–6½ gr.	2–8½ gr.	3–12 gr.
0.03–0.12 gm.	0.065–0.29 gm.	0.12–0.39 gm.	0.12–0.51 gm.	0.18–0.72 gm.
½–2 gr.	¾–4½ gr.	1–6½ gr.	1½–8½ gr.	2–12 gr.
0.03–0.12 gm.	0.048–0.29 gm.	0.065–0.39 gm.	0.09–0.51 gm.	0.12–0.72 gm.
½–1⅗ gr.	¾–3½ gr.	1–5 gr.	1½–6½ gr.	2–9 gr.
0.03–0.01 gm.	0.048–0.21 gm.	0.065–0.3 gm.	0.09–0.39 gm.	0.12–0.54 gm.
⅕–½ gr.	⅓–1 gr.	½–2 gr.	¾–2 gr.	1–3 gr.
0.013–0.03 gm.	0.02–0.065 gm.	0.03–0.12 gm.	0.048–0.12 gm.	0.065–0.18 gm.
¾–2 gr.	1½–4½ gr.	2¼–6½ gr.	3–8½ gr.	4–12 gr.
0.048–0.12 gm.	0.09–0.29 gm.	0.136–0.39 gm.	0.18–0.51 gm.	0.26–0.72 gm.
⅕–1⅙ gr.	⅓–2½ gr.	½–3½ gr.	¾–4½ gr.	1–6 gr.
0.013–0.65 gm.	0.02–0.15 gm.	0.03–0.21 gm.	0.048–0.29 gm.	0.065–0.36 gm.
⅕–¾ gr.	⅓–1½ gr.	½–2 gr.	¾–3 gr.	1–4 gr.
0.013–0.048 gm.	0.02–0.09 gm.	0.03–0.136 gm.	0.048–0.18 gm.	0.065–0.26 gm.
2½–10 m.	5–20 m.	7–30 m.	9–40 m.	12–50 m.
0.15–0.65 mil.	0.3–1.3 mil.	0.42–2.0 mil.	0.54–2.4 mil.	0.72–3.0 mil.
1/12–½ gr.	⅙–1 gr.	¼–2 gr.	⅓–2 gr.	½–3 gr.
0.005–0.03 gm.	0.01–0.065 gm.	0.016–0.12 gm.	0.02–0.12 gm.	0.03–0.18 gm.
1/26–1/12 gr.	1/13–⅙ gr.	⅛–¼ gr.	⅐–⅓ gr.	⅕–½ gr.
0.0025–0.005 gm.	0.005–0.01 gm.	0.008–0.016 gm.	0.009–0.02 gm.	0.013–0.03 gm.
1/26–1/12 gr.	1/13–⅙ gr.	⅛–¼ gr.	⅐–⅓ gr.	⅕–½ gr.
0.0025–0.005 gm.	0.005–0.01 gm.	0.008–0.016 gm.	0.009–0.02 gm.	0.013–0.03 gm.
1/26–½ gr.	1/13–¾ gr.	⅛–1 gr.	⅐–1½ gr.	⅕–2 gr.
0.0025–0.03 gm.	0.005–0.048 gm.	0.008–0.065 gm.	0.009–0.09 gm.	0.013–0.12 gm.
2½–5 m.	5–10 m.	7½–15 m.	9–18 m.	12–25 m.
0.15–0.3 mil.	0.3–0.6 mil.	0.42–1 mil.	0.54–1.08 mil.	0.72–1.5 mil.
1–2 gr.	2–4 gr.	3–6 gr.	4–8 gr.	6–12 gr.
0.065–0.13 gm.	0.13–0.26 gm.	0.18–0.36 gm.	0.26–0.52 gm.	0.36–0.72 gm.

TABLE OF DRUGS AND DOSAGE

Name	*Solubility of one part*		*Adult dose*
	In water At 77°F. At 25°C.	*In alcohol At 77°F. At 25°C.*	
Rhubarb and Soda Mixture	....	....	1–2 dr. 4–8 mil.
Rhubarb, Powder (as laxative)	ins.	ins.	5–10 gr. 0.32–0.65 gm.
Rhubarb, Powder (as purgative)	ins.	ins.	20–30 gr. 1.3–2.0 gm.
Rhubarb, Syrup	....	....	2–3 dr. 7.5–11.25 mil.
Rhubarb, Syr. Aromat	....	....	1–3 dr. 3.75–11.25 mil.
Rochelle Salt (see Acid, tartaric):			
Saccharin	0.5	170	3 gr.–5 gr. 0.2–0.32 gm.
Saccharum lactis	4.9	alm. ins.	6 dr.–6 oz. 23.5–185 gm.
Salicin	23.5	88.5	10–30 gr. 0.65–20 gm.
Salicylic acid (see Acid, salicylic):			
Salol (see Acid, salicylic):			
Salophen	ins.	s.	10–15 gr. 0.65–1 gm.
Salvarsan	s.	ins.	0.6 gm. or 0.01 gm. for every kilogram (2.2 lb.) of body weight.
Neosalvarsan	s.	ins.	0.09 m. or 0.015 for every kilogram (2.2 lb.) of body weight.
Santonin	5300	43	2–4 gr. 0.13–0.26 gm.
Senna Syrup	....	....	1–4 dr. 3.75–15 mil.
Serum Antidiphtheric (See Antitoxin):			
Serum Antitetanic	....	....	Prophylactic dose 1500 units
Sodium Benzoate	1.8	61	15–30 gr. 1–2 gm.
Sodium Bicarbonate	10	ins.	10–60 gr. 0.65–3.9 gm.
Sodium Bromid	1.1	16	10–60 gr. 0.65–3.9 gm.
Sodium Iodid	0.55	2	5–30 gr. 0.32–2 gm.
Sodium Phosphate (as laxative)	2.7	ins.	1–4 dr. 3.9–15.5 gm.
Sodium Phosphate (as hepatic stimulant)	2.7	ins.	20–40 gr. 1.3–2.6 gm.
Sodium Phosphate, Effervescent	v. s.	s.	2–4 dr. 7.7–15.5 gm.
Sodium Sulphate (Glauber's salt)	1	ins.	2 dr.–1 oz. 7.7–310 gm.
Sodium Salicylate (see Acid, salicylic):			
Spartein sulphate	1.1	3	⅙–2 gr. 0.01–0.12 gm.
Spearmint, water of (see Mentha Virides water)	....	....	Q. S.
Squills, Syr.	....	....	½–1 dr. 1.8–3.75 mil.
Strontium Bromid	0.35	r. s.	15–30 gr. 1–2 gm.
Strophanthus, tincture of	....	....	3–8 m. 0.2–0.5 mil.
Strychnin	6420	136	1/60–1/15 gr. 0.0011–0.004 gm.
Sulphonal (not advised in the treatment of children)			
Sulphur Precipitated	ins.	alm. ins.	1–3 dr. 3.9–11.6 gm.
Sulphuric acid, Arom.	....	....	5–15 m. 0.32–1 mil.
Tannalbin (see Acid, tannic):			
Tannigen (see Acid, tannic):			
Tartar Emetic (see Acid, tartaric) (Potassium and Antimony tartrate).			
Terebene sp		3	2–8 m. 0.12–0.5 mil.
Terpin Hydrate	200	13	2–5 gr. 0.13–0.32 gm.
Theobromin sodiosalicylate (see Acid, salicylic):			
Thymol (Anthelmintic)	1010	1	2–15 gr. 0.13–1.0 gm.
Thyroid Extract (Thyroideum siccum) (Desiccated thyroid glands).	ins.	ins.	2–5 gr. 0.13–0.32 gm.
Tolu Syrup	....	....	1–4 dr. 3.75–15 gm.
Trional (not advised in the treatment of children)			
Urotropin (see Hexamethylenamin):			
Veronal (not advised in the treatment of children)			
Whisky (see Alcohol):			

FOR INTERNAL USE (*Continued*)

Six months or 15 *lb.* 6.8 *kg.*	*One year or* 22 *lb.* 10.8 *kg.*	18 *months or* 25 *lb.* 11.35 *kg.*	*Two years or* 28 *lb.* 12.75 *kg.*	*Three years or* 32 *lb.* 14.64 *kg.*
2½–5 m. 0.15–0.3 mil. ⅕–½ gr. 0.013–0.03 gm. ¾–1 gr. 0.026–0.065 gm. 5–7 m. 0.3–0.42 mil. 2½ -7 m. 0.15–0.42 mil.	5–10 m. 0.3–0.6 mil. ⅓–¾ gr. 0.02–0.048 gm. 1½–2 gr. 0.09–0.12 gm. 10–14 m. 0.65–0.84 mil. 5–14 m. 0.3–0.84 mil.	7½–15 m. 0.45–1.0 mils. ½–1 gr. 0.03–0.065 gm. 2–3 gr. 0.12–0.18 gm. 15–21 m. 1–1.26 mil. 7½–21 m. 0.45–1.26 mil.	9–20 m. 0.54–1.3 mil. ¾–1½ gr. 0.048–0.09 gm. 3–4 gr. 0.18–0.26 gm. 20–25 m. 1.3–1.5 mil. 9–25 m. 0.54–1.5 mil.	12–25 m. 0.72–1.5 mil. 1–2 gr. 0.065–0.12 gm. 4–6 gr. 0.26–0.36 gm. 25–37 m. 1.5–2.22 mil. 12–37 m. 0.72–2.22 mil.
One grain to the pint for sweetening.				
10 gr.–2 dr. 0.65–8 gm. ½–1 gr. 0.03–0.065 gm.	20 gr.–4 dr. 1.3–16 gm. ¾–2½ gr. 0.048–0.15 gm.	30 gr.–6 dr. 2.0–24 gm. 1–3½ gr. 0.065–0.21 gm.	50 gr.–1 oz. 3–30 gm. 1½–4½ gr. 0.09–0.29 gm.	90 gr.–1⅕ oz. 6–36 gm. 2–6 gr. 0.12–0.36 gm.
⅖–½ gr. 0.026–0.03 gm.	¾–1 gr. 0.048–0.065 gm.	1–1½ gr. 0.065–0.09 gm.	1½–2 gr. 0.09–0.12 gm.	2–3 gr. 0.12–0.18 gm.
0.01 gm. for every Kilogram (2.2 lb.) of body weight.				
0.015 gm. for every Kilogram (2.2 lb.) of body weight.				
1⁄12–⅙ gr. 0.005–0.01 gm. 2½–10 m. 0.15–0.65 mil.	⅙–⅓ gr. 0.01–0.02 gm. 5–20 m. 0.3–1.3 mil.	¼–½ gr. 0.016–0.03 gm. 7–30 m. 0.42–2.0 mil.	⅓–¾ gr. 0.029–0.048 gm. 9–40 m. 0.54–2.4 mil.	½–1 gr. 0.03–0.065 gm. 12–50 m. 0.72–3.0 mil.
700	1000	1500	1500	1500
½–1 gr. 0.03–0.065 gm. ½–2 gr. 0.03–0.12 gm. ½–2 gr. 0.03–0.12 gm. ⅕–1 gr. 0.013–0.065 gm. 2½–10 gr. 0.15–0.65 gm. ¾–1½ gr. 0.048–0.09 gm. 5–10 gr. 0.3–0.65 gm. 5–15 gr. 0.3–1 gm.	1–2¼ gr. 0.065–0.136 gm. ¾–4½ gr. 0.048–0.29 gm. ¾–4½ gr. 0.048–0.29 gm. ⅓–2¼ gr. 0.02–0.136 gm. 5–20 gr. 0.3–1.3 gm. 1½–3 gr. 0.09–0.18 gm. 10–20 gr. 0.6–1.3 gm. 10–35 gr. 0.65–2.1 gm.	1¾–3½ gr. 0.11–0.21 gm. 1–6½ gr. 0.065–0.39 gm. 1–6½ gr. 0.065–0.39 gm. ½–3½ gr. 0.03–0.21 gm. 7–30 gr. 0.42–2.0 gm. 2–4 gr. 0.136–0.26 gm. 15–30 gr. 1–2.0 gm. 15–45 gr. 1.0–2.7 gm.	2–4½ gr. 0.03–0.276 gm. 1½–8½ gr. 0.09–0.51 gm. 1½–8½ gr. 0.09–0.51 gm. ¾–4¼ gr. 0.048–0.276 gm. 9–40 gr. 0.54–2.4 gm. 3–5 gr. 0.18–0.3 gm. 18–40 gr. 1.08–2.4 gm. 20–70 gr. 1.03–4.2 gm.	3–6 gr. 0.18–0.26 gm. 2–12 gr. 0.12–0.72 gm. 2–12 gr. 0.12–0.72 gm. 1–6 gr. 0.065–0.36 gm. 12–50 gr. 0.72–3.0 gm. 4–8 gr. 0.26–0.48 gm. 25–50 gr. 1.5–3.0 gm. 25–96 gr. 1.5–5.70 gm.
1⁄150–1⁄12 gr. 0.0004–0.005 gm.	1⁄75–⅙ gr. 0.0008–0.01 gm.	1⁄50–¼ gr. 0.0012–0.016 gm.	1⁄40–⅓ gr. 0.0015–0.02 gm.	1⁄30–½ gr. 0.002–0.03 gm.
1–2 m. 0.065–0.12 mil. ½–1 gr. 0.03–0.065 gm. 1⁄10–⅓ m. 0.006–0.02 mil. 1⁄1500–1⁄350 gr. 0.00004–0.00017 gm.	2–4 m. 0.12–0.26 mil. 1–2¼ gr. 0.065–0.136 gm. ⅕–⅔ m. 0.013–0.04 mil. 1⁄750–1⁄175 gr. 0.00008–0.00034 gm.	3–6 m. 0.18–0.36 mil. 1¾–3½ gr. 0.11–0.21 gm. ⅓–1 m. 0.02–0.065 mil. 1⁄500–1⁄150 gr. 0.00012–0.0004 gm.	4–8 m. 0.24–0.48 mil. 2–4½ gr. 0.03–0.276 gm. ½–1¼ m. 0.03–0.08 mil. 1⁄400–1⁄100 gr. 0.00015–0.0006 gm.	6–12 m. 0.36–0.72 mil. 3–6 gr. 0.18–0.36 gm. ¾–1½ m. 0.048–0.09 mil. 1⁄300–1⁄75 gr. 0.0002–0.0008 gm.
2–7 gr. 0.12–0.42 gm. ⅕–½ m. 0.013–0.03 mil.	4–14 gr. 0.26–0.84 gm. ⅓–1 m. 0.02–0.065 mil.	6–21 gr. 0.36–1.26 gm. ½–1½ m. 0.03–0.09 mil.	8–25 gr. 0.48–1.5 gm. ¾–2 m. 0.048–0.12 mil.	12–37 gr. 0.72–2.22 gm. 1–3 m. 0.065–0.18 mil.
1⁄12–⅓ m. 0.005–0.02 mil. 1⁄12–⅕ gr. 0.005–0.012 gm.	⅙–⅔ m. 0.01–0.04 mil. ⅙–⅓ gr. 0.01–0.021 gm.	¼–1 m. 0.016–0.065 mil. ¼–½ gr. 0.016–0.03 gm.	⅓–1¼ m. 0.02–0.08 mil. ⅓–¾ gr. 0.02–0.048 gm.	½–1½ m. 0.03–0.09 mil. ½–1 gr. 0.03–0.065 gm.
1⁄12–½ gr. 0.005–0.03 gm. 1⁄12–⅕ gr. 0.005–0.013 gm. 2½–10 m. 0.15–0.65 mil.	⅙–1 gr. 0.01–0.065 gm. ⅙–⅓ gr. 0.01–0.02 gm. 5–20 m. 0.3–1.3 mil.	¼–1¾ gr. 0.016–0.11 gm. ¼–½ gr. 0.016–0.03 gm. 7–30 m. 0.42–2 mil.	⅓–2 gr. 0.02–0.03 gm. ⅓–¾ gr. 0.02–0.048 gm. 9–40 m. 0.54–2.4 mil.	½–3 gr. 0.03–0.18 gm. ½–1 gr. 0.03–0.065 gm. 12–50 m. 0.72–3.0 mil.

TABLE OF DRUGS AND DOSAGE FOR EXTERNAL USE

Acid, Boric (Boracic Acid)

Used in powder (sodium borate) solution and ointment.

Acid, Carbolic (Phenol)

Used in lotion and ointment, 1 to 2 per cent. for its anesthetic and antiseptic properties.

Acid, Nitric

Used as caustic (undiluted).

Acid, Salicylic

Used in lotions and ointments, 1 to 3 per cent.

Acid, Tannic

Used for astringent effect, for irrigations in dysentery, and in suppositories.

Adrenalin

Used locally for hemostatic effect in solution of from 1: 10,000 to 1: 1000.

Amyl Nitrit

3–5 m., 0.2–0.3 mil. To be placed on a handkerchief and inhaled in spasmodic conditions, particularly asthma.

Argyrol

Used for same purposes as silver nitrate. It has no caustic effect. 10 to 50 per cent. strength. 20 per cent. solution for eyes of the newly born. Not as efficacious as silver nitrate.

Aristol (Dithymoldiiodid)

Used as antiseptic dusting powder, full strength. In solution 10 per cent. in oil it is used in various skin diseases.

Balsam of Peru

Used as a dressing for wounds and ulcers, and for burns when mixed with six parts of castor oil.

Barium Sulphate

When clinically pure and specially prepared for roentgenographic work is used in the proportion of 1 oz. barium sulphate to 3 oz. of the meal. In adult work from 4 to 6 oz. are given at a meal. More commonly used now than bismuth subcarbonate (see Bismuth Subcarbonate).

Belladonna Plaster (Emplastrum Belladonnæ)

Used as anodyne.

Benzoin

The compound tincture is used as antiseptic and protective, full strength, also in lotions. Employed in steam inhalations, 2 dr. to one pint.

Beta-eucain (see Eucain).

Bismuth Subcarbonate

Employed in roentgenography to render opaque the gastro-intestinal canal, 1 oz. of the bismuth may be given to every 4 oz. of the meal (see Barium Sulphate).

Bismuth Subgallate

Used as antiseptic drying powder, full strength, and in ointment 10 to 20 per cent. strength.

Bismuth Subnitrate

Used as antiseptic drying powder, full strength, and in ointment 10 to 20 per cent. strength.

Boracic Acid (see Acid, Boric).

Borax (Sodium Borate)

Used as very mild alkaline antiseptic wash.

Black Wash (Lotio Hydragygri Nigra)

Used as a soothing and antiseptic application in scrofulous and syphilitic ulcers.

Buchu, Infusion

Employed by bowel as diuretic (1 oz. leaves to one pint water). Adult dose 2 to 4 oz., 2.0 to 3.9 mil.

Cacao Butter (Oleum Theobromatis)

Employed in cosmetic ointments and in preparing suppositories. Is an emollient especially good for chapped skin (hands and lips). It melts at from 86 to 95°F. (30 to 35°C.).

Calamine (Zinc Carbonate)

Used as drying powder and in lotions and ointments. It is astringent and desiccant.

Calcium Liquor (Lime Water)

Used for its mild antacid and astringent properties in lotions for various skin affections.

Calcium Sulphid (Crude)

Used for various parasitic skin diseases in solutions of from 4 to 6 per cent. It is a depilatory when mixed with enough water to make a paste. It is an irritant.

Calomel

Used as antiseptic dusting powder, particularly useful in syphilitic lesions.

Camphor, Spirits

Used in liniments.

Camphor, Liniment (Camphorated Oil)

Used as a counterirritant. Not to be used for hypodermic administration.

Cantharides

Used as vesicant in the form of the cerate, plaster or collodion of cantharides.

Carbonic Acid Snow

Causes freezing of the skin and cessation of the functions. In this way it produces local anesthesia and arrests the local flow of blood. It is employed in the destruction of warts, moles, and vascular nevi.

Carron Oil (Linimentum Calcis)

Useful and soothing application in recent burns and scalds. Consists of equal parts of lime water and linseed oil.

Castor Oil

Used for its emollient effects in seborrhea and other skin diseases, also used as an enema.

Chalk, Prepared

Used as stimulating drying powder.

Chloroform

Used as vesicant. By inhalation as a general anesthetic.

Chrysarobin

Used either in 5 per cent. ointment, 1 to 2 dr. dissolved in chloroform, 1 oz., or preferably 1 dr. to 1 oz. of collodion, in the treatment of psoriasis and eczema.

Cocain

Used for anesthetic effect when applied locally in 3 to 10 per cent. solution. Employed in ophthalmoscopy to dilate the pupils without paralysis of accommodation. Local anesthetic when used hypodermically. Used in 0.2 to 4 per cent. solution.

Cod-liver Oil

Used as nutrient inunction.

Collodion

Used as protective. Also as vehicle for medication to skin.

Collodion of Cantharides, vesicating collodion (60 per cent. cantharides).

Collodion with Ichthyol, protective antiseptic (10 to 20 per cent. ichthyol.)

Collodion of Iodoform, protective antiseptic (5 per cent. iodoform).

Collodion of Oil of Cade, in eczema and psoriasis (1 to 5 per cent. oil of cade).

Collodion of Salicylic Acid Compound, for removal of corns (10 per cent. salicylic acid).

Creosote

Used in various skin diseases in 1 to 10 per cent. solution, also in inhalations for upper respiratory and pulmonary affections.

Dermatol (see Bismuth Subgallate).

Digitalis Infusion

Deteriorates readily. May be given by high enema in cases of suppression of urine.

Epsom Salts (Magnesium Sulphate)

In saturated solutions, will relieve localized edema. It also possesses mild anesthetic and antiphlogistic properties.

Ether

Used by inhalation as a general anesthetic.

Ethyl Chlorid

Used as a local anesthetic and by inhalation as a general anesthetic

Eucain (Beta-eucain)

Used as local anesthetic for same purposes as cocain. Is less toxic. Strength of 1 to 5 per cent. for local anesthetic. Sterilizing by boiling does not affect it.

Formaldehyd

Used as disinfectant, antiseptic and deodorant, in solution 0.5 to 2 per cent. The gas is employed for disinfecting purposes.

Glycerin

Beneficial for its hygroscopic and emollient properteis.

Goose Oil

Used as nutrient inunction.

Grindelia (Fluidextract)

Used as poultice or in solution for its soothing properties. In thus poisoning the application of the fluid extract diluted in water is used.

Grindelia

Used in conjunction with stramonium leaves in the preparation of asthma powders and cigarettes.

Guaiac Tincture

Used as gargle, 1 dr. to 6 oz. of water. Especially good in tonsillitis.

Guaiacol

Used externally for anesthetic effect; in various skin diseases; and mixed with equal parts of glycerin for analgesic effect in joint inflammation.

Hydrogen Peroxide

Used as an antibacterial agent and as a means of cleansing infected wounds. It is useful in hemorrhage because of its styptic effect. Used also as gargle and mouth wash in 25 to 33.3 per cent. solution in water.

Ichthyol

Used as local alterative and anodyne in skin affections, particularly those deep seated, as for example: erysipelas. Employed in ointments and solutions 5 to 50 per cent. strength. Also employed for deep seated inflammations, as in adenitis.

Iodin, Tincture

Used as counterirritant and antiseptic.

Iodoform

Used as antiseptic and alterative. Very little used because of odor. Powder and ointment are forms usually employed. Also used in surgical dressing.

Kaolin (Fuller's Earth)

Used as absorbing powder particularly valuable in "weeping" or moist surfaces.

Lard (Adeps, Axungia)

Used in preparation of ointments and cerates.

Lanolin (Hydrous Wool Fat)

Used as an ointment base. May require thinning in cool weather, in warm weather it is particularly useful.

Lead and Opium

One ounce of opium to the pint of liquor plumbi subacetatis dilutus is an anodyne lotion.

Lycopodium

Used as an absorbent to excoriated surfaces, particularly those appearing in the folds.

Mercury (Bichloride of Mercury)

Used as antiseptic in strength of 1: 10,000 to 1: 1000.

Yellow Oxid of Mercury (Hydrargyri Oxidum Flavum)

Used in ophthalmology in ointments of 1 to 2 per cent. Used also in various skin diseases.

White Precipitate (Hydrargyri Ammoniated)

Used in parasitic skin diseases and is particularly efficacious in impetigo (Indian fire) in 2 per cent. ointment.

Mercurial Ointment (Hydrargyri Unguentum)

Used as inunction in the treatment of syphilis ½ to 1 dr. at a dose. May be mixed with equal parts of vaselin in winter and lanolin in summer.

Menthol

Used externally as analgesic, antipruritic, refrigerant and sedative. Is employed in oils for upper respiratory tract in strength of 0.4 to 2 per cent. Used also in ointments in 1 to 5 per cent.

Mustard

Used as counterirritant in the form of bath, oil, pack, paper, paste, plaster and powder.

Mustard Plaster (Emplastrum Sinapis)

Used as rubefacient. Nitrous Oxid

Nitrous Oxid

Used by inhalation as a general anesthetic.

Novocain

Employed as local anesthetic in same manner as cocain

Oil of Cade

Used in parasitic skin diseases. In ointments, soaps, or in full strength rubbed into the skin.

Oil of Turpentine (Spirits of Turpentine)

Used as rubefacient and counterirritant in the form of liniments or diluted with three parts of a bland oil, or as turpentine stupe over the abdomen. Used in certain skin affections and its vapors sometimes employed by inhalation with advantage. In the proportion of eight parts turpentine to one part olive oil it is an excellent counterirritant to the thorax in pneumonias. Its action should be watched. Olive oil is its antidote when applied to the skin.

Olive Oil
Used as protective to skin. Employed also as enema to soften feces and to lubricate rectum.

Petrolatum (Vaseline)
Used as bland neutral protective. Also as base for ointments.

Potassium Sulphurate (Sulphurated Potash)
Used either as a lotion (5 to 10 per cent.); an ointment (10 to 12 per cent.); or bath ½ dr. to each gallon of water in parasitic skin diseases.

Potassium Permanganate
Used as antiseptic and disinfectant.
For mucous surfaces 1: 4000, 1: 2000.
On superficial wounds and ulcers 1: 1000.
See also medicated baths.

Quassia
Used as the infusion, 2 oz. in a pint of water, by bowel as a safe and valuable injection for seat worms (Oxyuris vermicularis).

Resorcin
Used in the form of lotions 1 to 2 per cent., ointment 1 to 5 per cent., paste and powder or soaps. It is antiseptic and antipruretic. Useful in skin diseases.

Nitrate of Silver (Argenti Nitras)
Used for its antiseptic astringent effect, 1 to 50 per cent. strength and as stick for caustic effect. In 2 per cent. solution for the eyes of the newly born.

Soap Liniment
Used as anodyne and rubefacient embrocation.

Sodium Bicarbonate
Used as sedative in skin diseases and burns in varied strength up to saturated solution.

Sodium Chlorid
Used in the form of baths. As an emetic in strength of ½ oz. to 1 oz. of water. Also used as clyster 1 oz. to one pint of water, and in the form of normal salt solution (liquor sodii chloridi physiologicus). It is administered either intravenously, subcutaneously, (hypodermoclysis) or through the colon (enteroclysis, Murphy drip).

Sodium Hypochlorite
Used in saturated solution to allay the itching and pain and reduce swelling incident to insect bites.

Starch (Amylum)
Used as base for drying powders; also added to baths for effect on skin.

Stramonium
Inhalation of fumes from leaves used in asthma.
See Grindelia.

Sulphur (Sulphur Sublimatum)
Used in suspensions or in ointments 5 to 20 per cent. strength in parasitic skin diseases.

Talcum (Purificatum)
Used as protecting, soothing dusting powder. It is used as a base for toilet and infant powders.

Tar (Pix Liquida)
Used as ointment, as stimulating and antiseptic application in various skin diseases.

Tropacocain
Sometimes used as a local anesthetic injected like cocain, and also for spinal analgesia.

Witch Hazel (Hamamelis, Fluidextract)
Used as sedative application to external inflammations.

Zinc Oxid
Used in lotions, ointments and powders for its astringent and sedative properties.

Zinc Sulphate
Used externally. It is an active astringent, mild caustic or disinfectant. As a collyrium (1 gr. to 1 oz.) or as an injection, gargle or wash for ulcers (1 to 3 gr. to the ounce).

CHAPTER XVIII

HELIOTHERAPY

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Heliotherapy—the art of healing disease by means of the sun's rays, one of the oldest, if not the oldest of the physiotherapeutic measures employed by man—has gained the recognition it deserves only during the past 20 years. Slowly the value of solar energy in the prophylaxis and treatment of disease is gaining a place in medicine. I say "slowly" because we know that it has been employed for over 2,500 years. Herodotus, 431 B. C., mentions the sun-bath and recommends it for restoring muscular tone, also warns against its use in summer in very weak individuals, and advises that the head be covered during exposure. Even in pre-historic times the influence of the sun's rays was appreciated. Cave dwellings in the old world faced toward the east or south, and I have personally noted the same in some of our southwestern states. The Greeks used the sun-bath extensively, exposing themselves from the terraces of their houses. Excavations at the temple of Æsculapius at Epidaurus have shown a large gallery facing south and adjoining the hospital; there is every reason to believe that this was used for sun-baths. In Rome, the Solarium situated as a rule on the roof of the house was considered a part of almost every home. In Central America and along the western coast of America the Indians lying in the sand exposed their nude bodies to the sun for the cure of rheumatism and consumption. Heliotherapy, while thus well known as a therapeutic measure in olden times, became a lost art for many centuries, practically until the nineteenth century. Several French, Dutch and German publications appeared during the early part of the nineteenth century dealing with this subject, but not until 1845, when Bonnet published his results, did it attract attention. Bonnet, Ollier and Poncet employed heliotherapy for tuberculous joint affections in a manner similar to the method employed today. In 1902 Bernhard of Samaden, Switzerland, began treating poorly healing wounds, tuberculosis of the joints, bones and skin, and severe anemias by means of sun-baths, but he did not report his results until 1914. Rollier, an assistant to Professor Kocher, convinced that surgery was not the method of choice in the treatment of external tuberculosis, established the first sanitarium for the systematic treatment of so-called surgical tuberculosis by means of the sun's rays. At that time Rollier was not acquainted with the work of Bonnet, Ollier, and Poncet; neither did he know that Bernhard at Samaden was working along the same line. Rollier of Leysin and Bernhard deserve the credit for establishing heliotherapy as a therapeutic measure of value. We are indebted to them, especially to Rollier, for having given an impetus to much experimental work. They also contributed definite rules and indications and reports of results from this treatment. Finsen, by his great work, established many scientific facts in regard to light and its effect upon the body cell and micro-organisms.

It will be necessary to re-state certain facts about light and review some of the experimental work before discussing the practical application of solar energy. Light is a wave-like disturbance which passes out in all directions from a luminous object through an ethereal medium. Waves of light are not all of the same wave-length. The difference in length makes itself known to our eyes as color. When the sun's rays pass through a prism they are dispersed into several kinds of light. We call this the visible solar spectrum, in which it is customary to distinguish seven colors—red, orange, yellow, green, blue, indigo, and violet. The red are the longest light waves; the violet the shortest. Each light-ray has three properties: (1) Heat production, (2) Light production, (3) Chemical action. The red rays are principally heat-rays; the violet rays have principally a chemical action and are often called actinic rays. Light is most intense in the yellow. On both sides of the visible spectrum are large fields, invisible to the eye, called the invisible spectrum or area of ultra-red and ultra-violet rays.

Effect of Light on Bacteria.—Downe first demonstrated that sunlight retards and inhibits the growth of bacteria. Dieudonné found that direct sunlight killed typhoid bacilli in one and one-half hours, arc-light (800 candlepower) in eight hours, and incandescent light in 11 hours. Arc-light is rich in ultra-violet rays. This inhibitory action is principally due to the chemical rays. The infra-red rays also have decided bactericidal power. All rays possess this action to some degree. Ultra-violet rays are bactericidal to a depth of 1½ cm. and inhibit microscopic growth to a depth of 4 cm. They also act on the ferments and toxins.

Action of Light on the Human Body.—This varies according to the intensity of the light, the altitude, and the individual. Sunlight in passing through the atmosphere to the earth loses a great part of its energy, the loss depending upon the position of the sun, the density of the air strata traversed, and the amount of moisture and foreign matter in the air. The chemical rays show the greatest, the heat-rays the smallest loss, except that the infra-red rays pass freely only through dry and dust-free atmosphere. The light intensity in higher altitudes is much greater than in the lowlands, and this difference is accentuated in the winter time. The volume of the ultra-violet rays increases with the increase in altitude. The difference in the ultra-violet rays' intensity in higher altitudes in summer and winter is comparatively small while in the lowlands it is great. Only those rays can produce an effect which are absorbed. The different rays vary as to their absorbability and, therefore, in their action. By exposure to the air and sun the skin is once more allowed to assume functions which modern civilization and clothing have taken away from it. The skin is not only an organ of sense and elimination but also of absorption; it absorbs not only gases but also light-rays. As rays act chemically upon the substances by which they are absorbed, metabolism is altered or, in this instance, increased. The effect of light on the body is not yet fully understood, but we regard the following facts as established, namely—(1) increased pigmentation, (2) increased metabolism, (3) increase in number of erythrocytes, (4) increased growth of hair, (5) local hyperemia, (6) decrease in number of respirations, (7) increase in depth of individual respiratory acts, (8) fall of blood pressure, (9) stimulation of nervous system.

Under the influence of the ultra-violet rays, principally, pigmentation takes place over the portions of the body exposed. This may be preceded by an erythema of varying intensity. The pigmentation is characterized by an increase of the pigment granules in the epithelial cells as well as in an increase in the number of chromatophoric cells. These are probably epithelial cells which have migrated into the connective tissue. Some believe that this pigmentation transforms short-waved chemical rays into long-waved rays which have a deeper power of penetration. It is also possible that the increase in pigment is a protective process on the part of the body against the ultra-violet rays. This pigment absorbs the violet rays but not the red. Lenkei has shown that only about a hundredth part of the light falling on the body penetrates it 0.5 cm., but some rays reach a depth of from 5 to 6 cm. When light penetrates the skin, practically all the chemical rays are absorbed by the blood. When we consider that exposure to light causes a local hyperemia the amount of energy absorbed by the blood must be enormous. Von Schlaffer has shown that blood during exposure to light absorbs light energy which it can transfer in the dark to a photographic plate. He very pointedly remarks "Is it not possible that it can also surrender this accumulated energy to the internal organs, and thereby influence their function and possibly pathologic processes?"

Death of bacteria is not due to light alone, but also to the inflammatory reaction caused by insolation. Leukocytes, which are chemotaxic, pass out of the vessels in great numbers, assisting in the destruction of the microbe.

Sunlight, in contra-distinction to antiseptics, does not damage body-tissues; it markedly increases cell-function.

Technic of Heliotherapy.—When employing heliotherapy it is of the utmost importance that we follow a proper technic. Thanks to Rollier, we are able to do this. After 15 years of experience and of careful observation, Rollier has seldom found it advisable to deviate from the rules he laid down many years ago. It is well worth remarking that Rollier, the man who has had the greatest experience in the treatment of external tuberculosis by means of solar energy and who has convinced many of the greatest surgeons of the world of the efficacy of his method, was himself a trained surgeon. He had ample opportunity at Professor Kocher's clinic to see these cases of external tuberculosis treated surgically by a master in that branch of medicine. Today he and practically all who have had an opportunity of giving heliotherapy a fair trial are convinced that surgery is not and will not be the method of choice in these cases. Operation does not change the constitution of the individual, except as it is influenced by the removal of one or more foci of infection. These foci, however, in the great majority of instances are merely local manifestations of the existing disease. If we are to cure the patient, if we are to prevent recurrences, we must aim to overcome the disease as a whole and not only the visible manifestations. In other words, we must increase the resistance of the individual and thus help him to combat the disease himself, at least until science gives us some specific remedy. The early French surgeons, even Bernhard of Samaden, relied principally upon exposures of the parts affected. Rollier considered this an incomplete procedure. From the beginning he considered a general exposure of the body as fundamental, local exposures as secondary in importance. According to our present-day conception of the disease, this is certainly the rational view. It follows that permanent casts and irremovable appliances, covering as they frequently do large portions of the body, must interfere with the proper application of heliotherapy. In order to get the best results we should endeavor to practise this method in places where in addition to the sun the patient may get the beneficial effects of an invigorating climate and air, that is at a moderate altitude in mountains. The dry invigorating air of the higher altitudes increases the action of the skin, dries up wounds when present, and helps to improve the general tone of the body. The treatment is really a combination of light and fresh air.

Patients on arriving at the sanatorium are put to bed for a few days, even those whose local condition does not make this imperative. They are thus allowed to become acclimated to the altitude. After three to five days, as a rule, they are moved to the verandas. After three to ten days the patient receives his first sun exposure. The head is covered with a white hat or an umbrella, the eyes protected with dark glasses and a white garment worn over the body. On the first day the feet are exposed three times for five minutes; on the second day three times for 10 minutes, and the legs to the knees three times five minutes; on the third day the exposures are increased five minutes; on the fourth day thighs are included; on the fifth day the arms; on the sixth day the back and on the seventh the abdomen and chest, so that at the end of the first week some parts of the body are exposed for one and one-half hours per day. The exposures are increased five minutes three times daily until the patient gets a full sun-bath for from three to five hours daily. This is the maximum, as a rule, although some bear the treatment for seven hours. Of course individualization is necessary, and in some instances the scheme of exposures must be modified to suit the case. Pulse and temperature are closely watched and used as an indicator of the patient's tolerance.

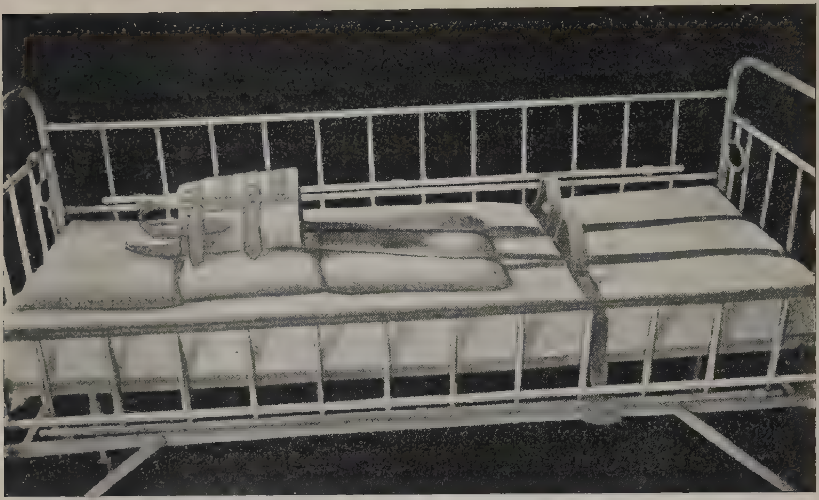

Fig. 112.—View of bed as arranged for immobilization of patient in recumbent position. (Rollier.)

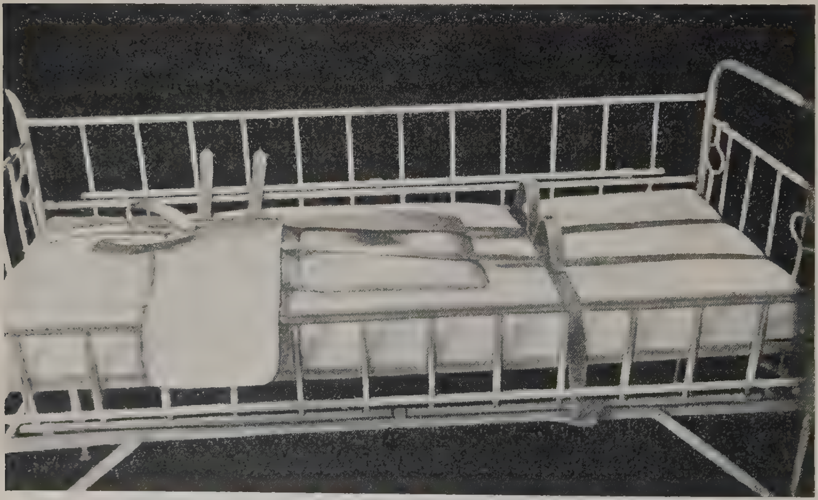

Fig. 113.—Same as Fig. 112 showing the shoulder straps to prevent the patient turning in bed. The three pillows are necessary to maintain the spine in the correct position. The upper one supports the shoulders and head, the middle one, convex, conforms to the spine and the lower one supports the pelvis. (Rollier.)

Those who have a limited experience with heliotherapy should not modify Rollier's directions. In certain localities and under certain conditions I believe we can begin the treatment with short complete exposures. This is particularly true in young children. I have repeatedly exposed them completely from the very start, but if I do so, I begin with a one- or two-minute exposure and only very gradually

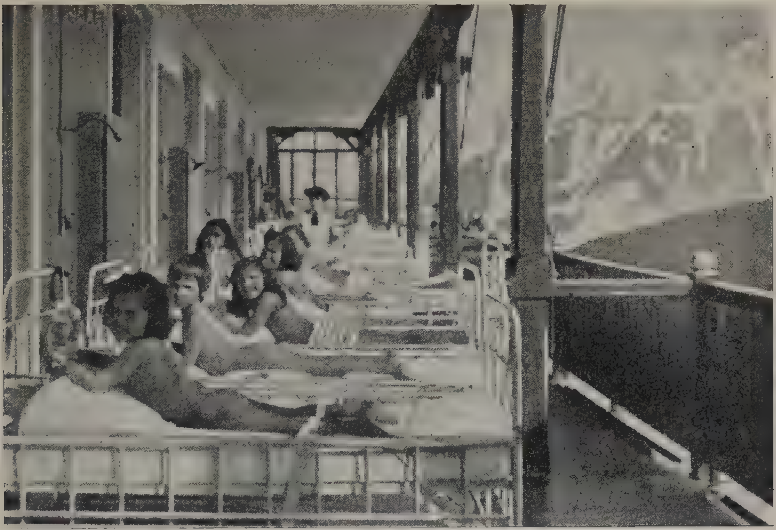

Fig. 114.—Immobilization in prone position with extension of thorax to correct the gibbus by means of progressive increase of the lordosis, at the same time providing for exposure of the back of the body. (Rollier.)

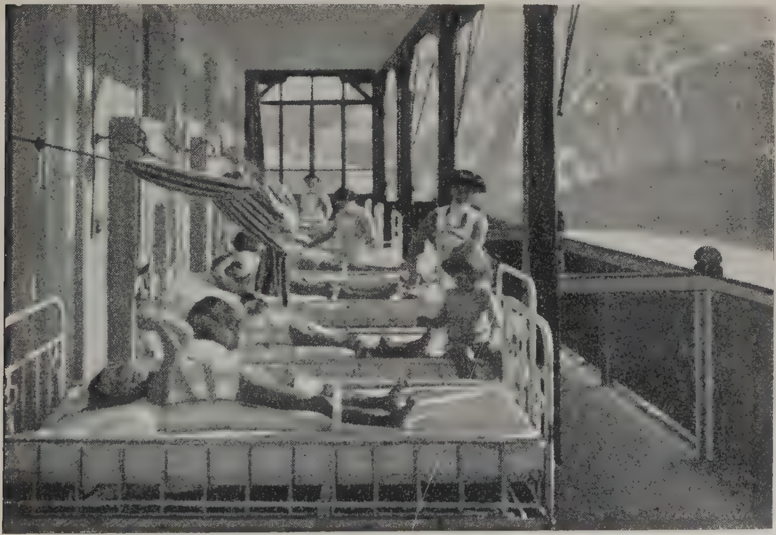

Fig. 115.—The same patients in dorsal position. It is only necessary to open the cloth corset to expose the anterior part of the body. (Rollier.)

increase the time. In anemias, in some cases of enlarged bronchial glands, in rickets and in cases where we are giving the treatment as a general tonic we may proceed in this manner. In tuberculosis of the lung it should not be attempted.

By the progressive increase of the time of exposure we avoid an erythema solare, and gradually the body acquires the desired degree of pigmentation which seems to be so essential. The rapidity and degree of pigmentation are, as a rule, in direct proportion to the power of resistance of the patient. Blonds, especially red blonds who pigment less, seem to offer a more unfavorable prognosis.

Nervous patients must be treated carefully as they often complain of headaches and nervous symptoms. These restless, excitable patients receive more air-baths and shorter exposures to the sun while the fat phlegmatic patients generally bear the sun-baths well.

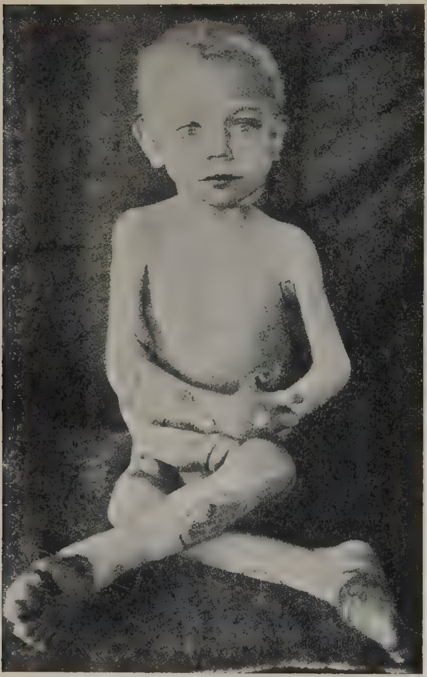

Fig. 116.—Patient on arrival at sanitarium. (Rollier.)

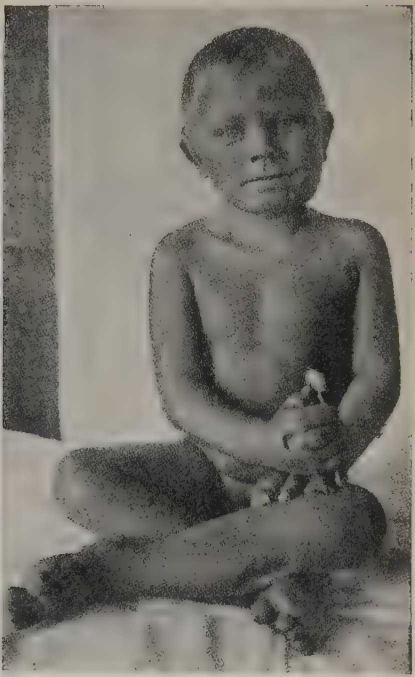

Fig. 117.—Same patient after treatment. Cured. (Rollier.)

As the healing of surgical tuberculosis requires rest of the parts affected, all cases except those of the upper extremities and lymph-nodes must be treated in bed. We must immobilize the parts. In the case of the upper extremities this is done with splints and bandages, which are removed during the exposure period.

Cases of tuberculosis of the hip, spine, lower extremities and peritoneum require rest in bed until healed, that is, until long after all pain has ceased and roentgenoscopy shows that the focus is healed. However, this requires months and often a year or more. It is in cases of long duration, particularly, that we observe the better effects of treatment carried on in high altitudes, for owing to the invigorating action of the mountain air they develop a good appetite and with a good diet gain in weight. In addition, increased tonus of the tissues soon develops, especially in children. At lower altitudes loss of appetite and the depressing action of the heat interfere with the systematic treatment.

Rollier has discarded the use of all non-removable appliances for immobilization as antagonistic to the principles of heliotherapy. By their use, no matter how skillfully made, the activity of the skin is greatly reduced, exposure to sun and air is impossible, metabolism decreases, and anemia and atrophy of the local tendons and muscles results. These factors tend to prevent healing, and if healing does take place, the functional result in many cases is poor. All appliances for immobilization are, therefore, removed daily during the period of exposure, except occasionally in the case of very refractory children with spondylitis. In these cases Rollier cuts large fenestra into the jackets. In general he applies extension apparatuses, splints, bandages and removable corsets of celluloid. In many cases of spondylitis, he employs a corset or jacket made of heavy material, which is in turn fixed to the mattress. Ambulatory appliances are used only after the tuberculosis is healed to protect the newly formed bone. These are usually made of celluloid so that they can be removed and treatment continued at home.

Medication is rarely employed; children, however, receive cod-liver oil.

Cold abscesses are given ample time to become absorbed unless they produce mechanical interference. Absorption occurs in a fair percentage of cases. Under all circumstances the rupture of an abscess is to be prevented because a mixed infection is practically unavoidable and makes the prognosis so much worse. If necessary, it is aspirated and injected with iodoform, 10 per cent. in oil or glycerin, to guard against mixed infection. This is done even daily if necessary. Every effort is made to avoid spontaneous rupture. If the patient has or develops a sinus, it is covered with an alcohol dressing when not exposed to the sun, to prevent infection as much as possible. No matter where the sinus is located, it shows an increased secretion when first exposed to light and gradually becomes dry. The granulations become healthy, and complete healing is frequently seen. According to Bernhard, the scars resulting from heliotherapy are firmer and significantly more elastic. Consequently they produce less contraction.

The high fever due to mixed infection drops; large open wounds and ulcerations heal with a minimum of deformity, and thickening of joint capsules and exudates are likewise absorbed in many cases.

Franzoni, an assistant of Rollier, has published an interesting article on the behavior of bone sequestra under the influence of light. As a rule from three to five months are required for a sequestrum to form and free itself from the surrounding tissues. Heliotherapy increases the speed of evolution of the sequestrum and causes its spontaneous expulsion. Rapid and firm healing follows. By this method, only the necessary portion of bone is eliminated, and no new wound surfaces are created, as in the case of sequestrotomy. Franzoni has observed that when cloudy days intervene a sequestrum near the surface retracts into the cavity only to be fully expelled after further exposure to the sun.

The analgesic effect of the sun's rays is seen in almost all cases. In rare cases the symptoms become exacerbated but soon abate; and all cases show a marked lessening and more frequently an absence of pain after a short period of treatment. This, in turn, helps the patient by allowing him plenty of rest and sleep, the exposure to the sun being conducive to sleep.

In many cases of joint tuberculosis we find a far better functional result after the use of heliotherapy than experience with other methods would lead one to expect. This is characteristic of the use of the sun and air treatment. Passive and active movements are not employed.

Bardenheuer, an assistant of Rollier, gives the following data from a study of the blood.

During from the the first to the third day of treatment, the red corpuscles usually increase about 500,000, and the white 3,000. The maximum increase is reached about the third day. The leukocyte count in closed cases runs about 10,000 and in cases with mixed infection from 13,000 to 17,000. He has found that cases in which the maximum increase in erythrocytes requires much more than three days offer a poorer prognosis. When healing starts there is a slight decrease in the number of erythrocytes.

Indications for Heliotherapy.—(1) So-called surgical tuberculosis. Here we must quote from Rollier's address in Munster in 1912:

"It is in surgical tuberculosis that we have seen the best results from heliotherapy, and we have made the treatment of it our life work. As a result of my experience in the use of the light cure in higher altitudes based on an experience of nine years, I maintain, today, that the cure of surgical tuberculosis in all of its forms, in all stages, as well as at every age of life, can be accomplished. The closed surgical tuberculosis always heals if one will only be patient, and above all, if one understands how to keep it closed. To transform a closed tuberculosis into an open one means to increase the gravity of the case a hundredfold. A diminution of the vitality of the tissues is the inevitable consequence."

2. Other forms of tuberculosis, as tuberculosis of the lung, bronchial gland tuberculosis, tuberculosis of the eye, tubercular pleurisy, tubercular adenitis, tuberculosis of the mucous membrane, etc.
3. Wounds healing poorly as in compound fractures, after mastoid operations, with bad infections, and from crushing injuries.
4. Skin lesions, such as ulcers, intertrigo, some cases of eczema.
5. Primary and secondary anemias of childhood.

6. Infantilism and debility of unknown origin.
7. Rickets.

The different forms of surgical tuberculosis, such as spondylitis, coxitis, tuberculosis of the peritoneum, and genito-urinary tuberculosis, all require, as a rule, from one to two or three years of continuous treatment. If this is done we see many beautiful functional results. We see tumor masses in the abdomen disappear, and if any scars result, they are usually less disfiguring than after operation. Enlarged glands gradually disappear spontaneously. Sinuses dry up and heal quite rapidly, leaving in many instances a soft, non-adherent, movable scar. In tuberculosis of the bronchial glands the common and frequent form in childhood, we consider it a necessary treatment for two reasons. The active cases are soon benefited and later cured; and latent cases are spared the terrible sequelæ resulting from the spreading of the disease to the adjacent tissues and through the entire body by an increase in their resistance.

In cases of tuberculosis of the lung special care must be employed. If we follow the directions of Rollier to the letter, and choose cases in which the disease has become somewhat torpid and which have little, if any, temperature, we will avoid exacerbations, and will soon be convinced, in spite of unfavorable reports, that even in pulmonary tuberculosis heliotherapy has a field of application.

In cases of thickened pleura I have seen most gratifying results.

The anemias of childhood are benefited. Within a few weeks after we have started sun-exposures the mothers, almost without exception, remark about the increase in appetite and gain in weight. A marked change in the appearance of the child and improvement in the blood picture results.

The work of Alfred Hess has demonstrated the use of Heliotherapy, both as a prophylactic and curative agent in rickets. He again has called attention to the well known fact, that exposure through glass is almost useless. Glass absorbs a very large quantity of the chemical rays.

We are not trying to proclaim heliotherapy as a panacea for all the ailments of childhood, but we do believe that if it were employed more extensively much unnecessary medication and surgery would be avoided. While the results are better at a higher altitude, the treatment can be carried on anywhere. Hospitals should have verandas and sun-parlors. The flat roofs of houses and hospitals can frequently be utilized. It is at times a little more difficult to carry on the treatment in the home, but some place, as a rule, can be found that is sheltered. We have many times improvised a shelter in the garden or yard, making a cubical four or five feet high, and protecting the patient on two or three sides, as necessity demanded, by means of an ordinary piece of canvas. Even where we employ other methods of treatment in the conditions mentioned, heliotherapy in some form will help our little patients. The seashore, especially in the milder parts of our country, is quite a desirable place but less so than the mountains. Mountains at an altitude of 1000 to 8000 feet offer the most favorable surroundings because the air is often dry and certain districts are protected against the winds. There is less fog, less dust, greater intensity of light, greater volume of actinic rays and no intense summer heat. In addition, there are more days of sunshine. In many places the treatment is carried out during the entire year.

We believe infants should have a daily exposure of their bodies to the air and, if possible, to the sun as a general hygienic measure. In a large number of the many cases of nutritional disturbances, which the pediatrician has to deal with, a moderate use of the sun's energy will assist the child in its metabolic functions and hasten its recovery.

Now, just as we are rehabilitating our wounded soldiers, we should rehabilitate our little tuberculous patients. I believe this is best accomplished by the systematic use of heliotherapy because it aims to cure the disease and not merely treat one of the symptoms; and because it so often results in restoration of function to such a degree that the little individual will be self-supporting in later life and will not be dependent on the state or community.

CHAPTER XIX

DISEASES OF THE NEWBORN

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HEMORRHAGE AND HEMORRHAGIC DISEASE

A tendency to spontaneous and prolonged hemorrhages is often seen in the infant during the first few days after birth. The hemorrhages may be limited to one organ or part or may be very general in character involving the skin, mucous membranes, meninges, and the parenchymatous organs. Among those conditions which are most often described as the cause of various types and forms of hemorrhage in the newborn, we find traumatism occurring during the process of birth, asphyxia, sepsis neonatorum, congenital syphilis, hemophilia, gastric or duodenal ulcer, and more recently, a hemorrhagic diathesis.

Hemorrhage from traumatism at birth usually results from injury to the head by extreme pressure and distortion. The resulting localized hemorrhage is apt to be confined to the meninges and brain. Asphyxia evidently plays a role in producing a tendency to hemorrhage, but whether as the result of increased pressure causing injury to the walls of the blood-vessels, or as an actual change in the blood stream, remains to be determined. Sepsis is undoubtedly often accompanied by hemorrhages in the newborn, but it is in the same degree and manner that one would expect to find it in older children or adults. It is well recognized that infants affected with congenital syphilis have a tendency to hemorrhage which is often unusually severe. Hemophilia as a distinct entity in the newly born has been practically discarded. Gastric and duodenal ulcers are probably responsible for a not inconsiderable number of our so-called cases of melena, neonatorum. After taking into consideration all those cases of hemorrhage in the newborn which may be attributed to traumatism, asphyxia, sepsis, syphilis, gastric ulcer, etc., a very large percentage of cases of spontaneous hemorrhage still remain which can be explained in the light of our present knowledge only as the manifestation of a hemorrhagic diathesis, the underlying cause being not yet satisfactorily explained. The hemorrhage occurring in sepsis, traumatism and syphilis will be discussed here only as they may enter into the general picture of the etiology, symptomatology and treatment of hemorrhagic disease of the newborn.

Statistics setting forth the occurrence of hemorrhage in the newly born have been of little value because of the wide variation in statisticians' minds as to what exactly constituted a pathologic entity at necropsy. Because of this and the general confusion which exists as to the underlying cause of the condition, no clear classification has been made of cases specifically due to some such well-recognized entity as injury, sepsis, syphilis, ulcer, etc. For example: Statistics vary all the way from the report of the Lying-in-Asylum of Boston of 0.6 per cent. in 5225 cases, to the report of Epstein of 8 per cent. in 740 infants at the Foundling Asylum at Prague. Again, Weyhe reported 12 per cent. in 950 necropsies of the newborn; much later Warwick reported 50 per cent. in 36 cases, and Parrot 76 per cent. in 34 cases. More than 25 per cent. of Warwick's cases showed a picture of general multiple hemorrhage. Congenital syphilis plays a rôle, as statistics show that in from 10 to 15 per cent. of these cases hemorrhage will occur. It is an open question as to just how often sepsis is taken into consideration in statistical analyses. We do know, however, that so-called sepsis neonatorum is becoming increasingly rare. On the other hand, largely perhaps because of better means of diagnosis, the percentage of hemorrhagic disturbance in the newborn is assuming much larger proportions, more especially the multiple hemorrhage type which affects principally the visceral organs and membranes.

Etiology.—While the etiology in a certain large group of cases remains obscure, we can readily eliminate from discussion those due to direct traumatism or ulceration. Rodda's recent work has crystallized our method of approach to cases in which, in the newborn, the symptoms point to possible hemorrhage. He has shown that, if the bleeding and coagulation time fall within normal limits, intracranial hemorrhage may be ascribed to some purely local condition, such as a ruptured blood vessel, or in the case of so-called melena neonatorum, to some condition such as gastric ulcer, but not in either case to a state of hemorrhagic diathesis in the infant. He further demonstrates, by a study of a large number of newborns, that the coagulation and bleeding time is gradually prolonged in the normal child, reaching its heighth by the fourth day, and ordinarily returning to normal by the fifth or sixth day. This coincides with the fact that idiopathic hemorrhage ordinarily appears from the second to the fourth day after birth. He has shown that the coagulation and bleeding time are always very greatly prolonged in cases of idiopathic hemorrhage. For instance, in cases of true melena neonatorum, the coagulation time is often prolonged to 90 minutes, and the bleeding time to hours. Similarly in cases of cerebral hemorrhage, the coagulation time reaches 90 minutes, with the bleeding time proportionally prolonged. Therefore, when the results of these blood tests fall within normal limits, hemorrhagic disease as an entity may be excluded from the diagnosis, but when the results prove an undue prolongation, we may be dealing with either a primary hemorrhagic diathesis, or a secondary hemorrhagic status which may be due primarily to syphilis, or sepsis, in which case other clinical signs would very probably lead to a differentiation. In discussing the underlying factors involved in the production of a status hemorrhagicus in the newly born, one finds little in the literature from which to draw conclusions that are of value. As has been stated before, in nearly every discussion of the subject, the minds of the authors have been confused because of a lack of clear differentiation between cases of true hemorrhagic disease, and those in which the hemorrhage is the result of traumatism, etc. Primarily, we may now be certain of one fact, that in these cases there is some grave change in the blood formula of the newborn with failure of the blood to coagulate, and that the change in the walls of the blood-vessels is only of secondary importance. Proof of this lies in the fact that the introduction of whole adult blood into the blood stream of the hemorrhagic infant will in all cases materially inhibit, and in nearly all cases entirely control the bleeding, causing a coincident return to normal of the bleeding and coagulation time. It would be hard to believe that were the walls of the blood vessels primarily at fault, the introduction of this blood would in any way materially affect them. However, one can not say just what new elements enter the infant's blood stream with the mature blood. It may furnish the coagulating elements perhaps lacking in the blood of the child with hemorrhage or possibly it introduces certain ingredients which stimulate the production of coagulating elements in the child's blood, or again certain components of the adult blood may inhibit the action of those factors which are producing the abnormal condition of the infant blood. Just what the action is has not been satisfactorily determined, however, we recognize clinically that in idiopathic cases or those due to congenital syphilis, the patients are benefited to a marked degree, and in septic cases are temporarily benefited, by the introduction of adult blood. This argues that the underlying causes of hemorrhagic tendencies in these different clinical entities may be of the same nature. When seeking for a cause for hemorrhagic status in the newly born, one finds that the usual explanation is centered around those conditions which would produce abnormal stasis in the vascular system. This stasis might be the result of compression of the cord during labor, asphyxia, atelectasis, congenital anomalies of the circulatory system or viscera. It is just possible that any condition which would materially interrupt the normal flow of the blood stream in the newborn during or immediately after birth, or interfere with the normal physiologic interchange of its chemical elements, might produce more or less permanent variation from the normal equilibrium, and be responsible for a hemorrhagic status, or the abnormal condition of the blood stream itself may in some way interfere with the nerve control of the blood-vessel walls. Much discussion is found in the old literature as to whether or not melena neonatorum could be the cause of cerebral hemorrhage, but with the modern conception of a status hemorrhagicus little doubt exists that these two conditions occur coincidently. In considering the relation of asphyxia neonatorum to hemorrhagic disease, we know that children who are deeply asphyxiated at birth are apt to show evidence of hemorrhage, especially of the cerebral type, but whether this is secondary to the asphyxia or to the long-continued pressure which produced the asphyxia, has not as yet been determined. It is also possible that the trauma incident to the passage of the head through the birth canal may be the exciting factor in producing the hemorrhagic status. If it could be proved that interference with the circulation could bring about an interference with the normal coagulation time of the blood, it would be easy to imagine ample opportunity for the interference with the circulation in the cord during labor. A discussion of the etiology of hemorrhagic disease in the newborn cannot be dismissed without mentioning, in passing, as has been noted by several authors, that in certain instances the disease has been almost epidemic in certain institutions. This of course would point to an infectious origin. It might be suggested in this connection that the beneficial action of the adult blood might be due to the introduction of specific antibodies, rather than to any direct stimulation of coagulating elements. Undoubtedly, the accurate study of the coagulating and bleeding time of the newborn under all conditions will accumulate evidence which will do much to clarify the situation in the future.

Pathology.—In many children dying of hemorrhagic disease, one finds little except evidence of the draining of blood from the various organs and tissues, while in other cases evidence of local hemorrhages will be present throughout most of the organs of the body. As has been noted in Warwick's series, cerebral hemorrhages were present in 50 per cent. and generalized hemorrhages in 25 per cent. Cerebral hemorrhages are most apt to occur in the meninges and are ordinarily diffuse in character. They are usually located over the cerebellum, or at the base. But what is very important is the fact that minute hemorrhages are apt to be found within the brain substance. Areas of hemorrhage are usually found in the lung tissue and upon the pleural surface, with occasional extravasations of blood into the pleural cavity. Ecchymoses often occur in the pericardium. The thymus gland and the suprarenal capsules frequently contain small extravasations. Evidence of hemorrhage is usually found both on the surface and in the substance of the liver, the spleen, and the kidneys. Although quantities of blood are frequently found in the intestinal tract, little change is noted in the intestinal wall except when ulceration is present. In true cases of hemorrhagic disease, little evidence of change appears in the walls of the blood-vessels, and even in the presence of rather massive hemorrhage, a ruptured blood-vessel is rarely discovered. Therefore, in cases of idiopathic hemorrhage in the newborn, little structural tissue change occurs, the picture being merely that of extravasation of blood, which has undergone a greater or less degree of decomposition, depending upon how long before death the hemorrhage took place.

Symptoms.—The one constant symptom in cases of true hemorrhagic disease of the newborn is the prolongation of the coagulation and bleeding time. This symptom is often present for some hours before any actual bleeding occurs, and is always present during the period of hemorrhage. Status hemorrhagicus usually develops from the first to the fourth day, and occurrence is little influenced by the previous condition of the infant. It is possibly more apt to occur in infants born asphyxiated, but may develop in any child regardless of the duration or severity of labor. In true hemorrhagic cases, the picture varies in accordance with the site of the predominating hemorrhage. In most cases, nothing unusual occurs to attract the attention of the clinician until the hemorrhage begins. This may be blood oozing from the umbilicus or skin, a bleeding from the mucous membranes of the mouth, a vomiting of blood, the passing of bloody or tarry stools, or the sudden appearance, with rapid and continuous increase in size, of a cephalhematoma. When one or more of these symptoms occur and the coagulation and bleeding time is found to be materially prolonged, the diagnosis becomes at once apparent. However, when there is no external or superficial evidence of the bleeding, as in cerebral hemorrhage or an extravasation into the lung or other organ, the diagnosis presents a more complex problem. No characteristic variation in temperature may be attributed to this condition. Sometimes a rather high fever is manifest, but in most cases the temperature is normal, subnormal or fluctuating. Icterus, which has been advanced as symptomatic by some authors, is of no importance, as Rodda and others have shown that in physiologic icterus neonatorum, the bleeding and coagulation periods are normal. In hemorrhagic disease, there is a marked pallor which becomes profound if the bleeding continues, and the child becomes exsanguinated. This occurs more rapidly in cases in which melena is the predominating symptom. In this connection however it must be remembered that while the apparent bleeding may only be a slight oozing from the umbilicus or a skin abrasion, in all probability other hemorrhages are occurring internally at the same time. Emesis resulting from hemorrhages into the stomach is usually dark brown and the quantity is small. Blood passed from the bowel is ordinarily very dark, but causes a red stain on the napkin. It is as a rule not well clotted, and is intimately mixed with meconium. The amount of the blood vomited or passed by the bowel is not always in proportion to the amount of hemorrhage taking place, as frequently, at necropsy, large quantities of blood are found in the stomach and intestines, when only small amounts have been vomited or discharged from the bowel. When the hemorrhage is gastro-intestinal or superficial in type, thus causing no disturbance of function of the nervous system or other organs, the picture is one of profound anemia, great prostration and failure to nurse, persisting in severe cases until the infant is completely exsanguinated and death ensues.

When the predominating hemorrhage is cerebral in type, the picture is similar to that resulting from injury to the blood-vessels in the same region. The infant presents a picture of distress. There is usually a rather characteristic, feeble, plaintive cry, failure to nurse, and variable temperature. Convulsions are common and opisthotonos is often present. Spasticity of one or more extremities usually exists, twitching of the arms, legs and face, and often cyanosis.

Respiration is usually disturbed—it may be very rapid, very slow or irregular, the heart is slow and the pulse feeble. Nystagmus is occasionally present and the pupils are usually contracted, fixed and possibly unequal in size. In cases of massive hemorrhage, there is increased tension and bulging of the fontanelle. Spinal puncture very often discloses a bloody spinal fluid. Death may occur at any time up to the third or fourth day in these severe cases. In milder instances, only slight convulsive twitchings of one or more of the extremities, or possibly one or more generalized convulsions may be noted. However, many times such mild hemorrhages are responsible for the spastic paralysis of early childhood. This is especially true in cases in which the bleeding consists of minute hemorrhages into the cortical substance of the brain.

In cases in which the predominating hemorrhage is in the lungs, symptoms occur similar to those described under cerebral hemorrhage, such as apprehensive cry, failure to nurse, irregular breathing, cyanosis, and occasionally opisthotonos and convulsions, but there is an absence of the rigidity or paralysis of the extremities, the fixed pupils and the bulging fontanelle. Occasionally, massive hemorrhages occur into the suprarenal capsules which may be felt as tumor masses in the region of the kidney. At other times, blood may appear in the urine, indicating hemorrhage occurring in the kidneys.

Diagnosis.—Conditions to be differentiated from hemorrhagic disease of the newborn are congenital syphilis, sepsis neonatorum, gastric and duodenal ulcer, traumatic cerebral hemorrhage, hemorrhage from the ears, eyes, nose and mouth, and hematuria from other causes. In syphilis, hemorrhage usually does not appear as early as in true hemorrhagic cases. There is usually an enlargement of the spleen and liver, often a positive Wassermann, history of syphilis in the parents, and often a bloody mucus discharge from the nose. In sepsis neonatorum, we have the history of infection, the general symptomatology of sepsis existing usually for days before signs of hemorrhage appear. The spleen and liver are usually enlarged, the hemorrhage showing as purpuric manifestations in the skin and mucous membranes, an oozing from the umbilicus, or as in the case of hematuria, presenting evidence of nephritis or cystitis. The bleeding and coagulation time may or may not be normal. In gastric or duodenal ulcer, we have the exact picture of melena neonatorum, but the bleeding and coagulation time will not be prolonged. In the case of traumatic cerebral hemorrhage, usually a history exists of marked distortion of the bones of the head from the severity of the birth process. The normal bleeding and coagulation time, the more rapid progress of the hemorrhage which often causes death within the first 24 hours, and the absence of hemorrhages in other parts of the body, will help to make a differential diagnosis. A hemorrhage occurring from the ears usually results from an otitis; if from the eyes it will be accompanied by a conjunctivitis. Hemorrhages occurring from the nose or mouth are of especial interest because the blood is very apt to be swallowed by the infant, to appear later in an emesis or in tarry stools. It is interesting to note in this connection that when this appearance of blood is apparently without explanation the source may often be found in the cracked and bleeding nipple of the mother. Hemorrhages from the nose and mouth are most frequently due to congenital syphilis, in which case, as is true in hemorrhagic disease, the bleeding and coagulating time might be definitely prolonged. If the hemorrhages were due to local disturbances, or to blood coming from the mother's nipples, the bleeding and coagulation time would be normal. We must also remember that hematuria is in the newborn occasionally due to abrasions in the kidney or ureters from the passage of uric acid infarcts.

Treatment.—The treatment of hemorrhagic disease has become much simplified in the last few years. The introduction of whole adult blood either subcutaneously or intravenously is generally recognized as almost a specific remedy. In all cases of suspected hemorrhage bleeding and coagulation time should be routinely determined. The bleeding time may be ascertained by the method described by Duke. After cleansing the heel with alcohol or ether, a puncture about 0.5 cm. deep is made with a lance blade to ensure a free flow of blood. As fast as blood appears it is gently removed by touching with a piece of blotting paper. This is continued until blood ceases to appear from the wound. By this method the average normal time for the newborn is about three and one-half minutes, with a normal range of from two to five minutes. In cases of hemorrhagic disease the oozing sometimes continues for many hours or even days. The coagulation may be determined coincidently with the bleeding time from the same puncture. Rodda has developed a method which is very simple, accurate and easily applied either in a ward or in the home. The apparatus (Fig. 118) as described by Rodda, consists of a spring lance (a simple scalpel will suffice), two 1½-in. watch glasses and No. 6 lead shot, all of which are easily obtained and transported. Glass and shot should be cleansed, preferably by washing with soap and water followed by alcohol and ether. Needless to state, the lance should be sterile, which implies freedom from old blood. The heel of the infant is sponged with ether, a puncture is made with the lance

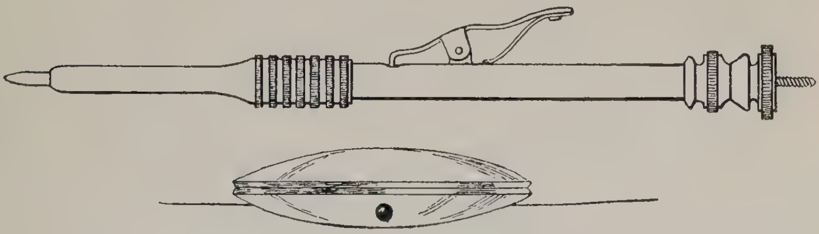

Fig. 118.—The apparatus used consists of a spring lance, two watch crystals and No. 6 lead shot. (Rodda.)

blade set (about 0.5 cm.) to produce a free flow of blood without the slightest pressure. A clean watch glass containing a No. 6 shot receives the second drop of blood. A second watch glass is inverted over the first. The watch glasses are gently tilted every 30 seconds until the shot no longer rolls, but is fixed in the clot. The end-result is sharply defined, the shot is firmly embedded, so that the glass may be inverted without dislodgment of the shot. At times, due to the forcing out of serum, the whole clot may move, carrying the shot with it. This is no source of confusion, for the shot will be found enmeshed in the fibrin and does not roll or rotate. A poorly formed clot or one showing slight retraction is patent, for in such instances the shot continues to roll.

The average coagulation time in the newborn with this method is seven minutes, with a normal range of from 5 to 9 or 10 minutes. In cases of hemorrhagic disease the clotting time is often delayed 30, 60, or even 90 minutes, the clot never being firm in these long delayed cases. It is advisable also to determine the hemoglobin while noting the clotting and bleeding time as this will give an idea of the amount of hemorrhage which has taken place. In severe cases recovery has occurred after the intravenous injection of whole blood when the hemoglobin reading has been 20 or below. As soon as a definitely prolonged bleeding or coagulation time has been obtained, it is advisable to begin the administration of whole blood. As usually there is not time to obtain a Wassermann reaction to insure the child against an infection with syphilis it is customary to use blood obtained from either parent. The fact is now well established that the blood of the newborn will absorb without hemolysis or agglutination the blood from a donor of any group, so the blood of the father or mother may be used with impunity for intravenous injections. In all cases treatment should be first by repeated subcutaneous injections of blood and only after failure to control the hemorrhage by these injections and when the case becomes grave are intravenous injections resorted to. For the purpose of subcutaneous injections, the uncitrated blood is used in order to preserve all the coagulating factors present. A 30 c.c. Luer syringe (a smaller one may be used) and a venepuncture needle are all that is required. These are sterilized in the usual way by boiling and are cooled to room temperature. It is often of advantage to have the syringe lubricated with vaseline, though this is not essential. A venepuncture is made in the usual way and without changing the needle the contents of the syringe are injected deeply into the subcutaneous tissues of the child. The injection may be made at any convenient point but the area of choice is probably the upper part of the back in approximately the midscapular line, the blood being injected outward toward the axilla. It is well to massage the area at the same time as it facilitates the injection and probably lessens the pain by distributing the blood over a wider area. After the needle is withdrawn and the area massaged, the opening is sealed with collodion on cotton. No especial skill is required for this procedure, the only requisite for success being to have everything in readiness before beginning so that the injection may be made quickly before clotting of the blood occurs. The blood is absorbed within a reasonable length of time, often in the course of a few hours.

Owing to the small size of the veins in the newborn, the superior longitudinal sinus at the posterior angle of the anterior fontanelle is the point usually selected for intravenous puncture when any quantity of blood or other fluid is to be introduced. For obvious reasons citrated blood should be used for this purpose. We are aware of no standardized technique for this procedure, each operator usually employing the method which he has found most successful. The method described below is one which we have frequently employed with satisfactory results and we know of none more simple yet equally safe.

The blood to be injected is collected as for any transfusion. A venepuncture is made in the arm of the donor with a needle of large caliber and the blood allowed to flow into a sterile glass graduate containing the amount of sodium citrate solution necessary for the amount of blood to be collected, (10 c.c. of a 3 per cent. solution of sodium citrate in freshly distilled water for each hundred cubic centimeters of blood). The container is then covered with a sterile cloth and set into a vessel containing water at about 105°F. to prevent cooling of the blood. For injection into the sinus the following apparatus is used: The puncture is made with a short spinalpuncture needle of small caliber (made by filing a needle of standard length). Upon this needle a guard is placed so that the depth of the puncture may be regulated. Numerous devices for this purpose have been described. A simple one, consisting of an ordinary brass set-screw can easily be made by any mechanic. For injection a 30 c.c. Luer syringe is employed. This is connected to the needle by means of a section of rubber tubing, about 6 or 8 in. in length. Into one end of this tube is inserted an ordinary metal "adapter" for the syringe, and into the other end an adapter to fit into the needle. These adapters are used to facilitate connection and disconnection of the various parts. The advantage of the rubber tube is that it permits almost unlimited manipulation of the syringe without incurring the risk of disturbing that portion of the needle in the sinus.

The child is securely wrapped in a blanket and the head firmly held, face upward, at the edge of the table in front of the operator. The area over the posterior portion of the fontanelle is prepared with iodin and alcohol (having been previously shaved if necessary). The guard on the needle is set at about three-eighths of an inch, the needle is inserted at an inclination of about 60° at the posterior angle of the fontanelle and the stilette is withdrawn. When the sinus has been satisfactorily entered there will be a ready flow of blood. When this occurs the assistant fits the adapter on the end of the rubber tube into the needle, the other end of the tube in turn being connected to the syringe, both having previously been filled with blood. The blood is then injected very slowly by exerting the very slightest pressure upon the plunger of the syringe which will be sufficient if the flow into the sinus is free.

While the injection is being made the child should be watched for any evidences of cortical irritation and the injection stopped if this becomes manifest. If more than the contents of one syringe is to be injected the syringe alone can be disconnected, the rubber tube pinched to prevent a reflow of blood, the syringe refilled, connected again and the injection continued. After withdrawing the needle from the sinus, the area is gently massaged in order to close the puncture wound which is later sealed in the usual way with collodion and cotton. Extravasation of blood under the scalp may often be prevented by keeping the child's head elevated for some time. In estimating the amount of blood to be injected at one time we have made it a rule to use not more than one-sixtieth of the body weight.

It is obvious that the injection can be more simply made by using a syringe attached to an ordinary venepuncture needle, but in making injections into the sinus one is at best proceeding blindly and all possible safeguards should be thrown about the process. This is especially true in the newborn in whom, because of the molding of the head, the exact location of the sinus cannot be mapped out with mathematical exactness. The gravity method of injection may be used instead of the syringe method, but if none but the slightest pressure is exerted, the latter is equally safe and much more efficient.

In cases of cerebral hemorrhage surgery offers a new procedure for permanent relief from pressure over the cortex, and subsequent paralysis, convulsions, etc. When the bleeding time has returned to normal, or, in the case of traumatic hemorrhage, when the bleeding time has not been prolonged, when we have definite evidence of pressure over the cortex, and the condition of the infant will permit, excellent results have been obtained by raising a bony flap and removing the clot. For best results this should be done at the earliest possible moment and surely within the first 10 days.

In all cases of hemorrhagic disease the general supportive measures recommended in other affections of the newborn, such as administration of breast milk and other fluids, the maintenance of body temperature and stimulation when necessary, should be carefully carried out.

Prognosis.—With the advent of whole blood therapy in cases of true hemorrhagic disease the prognosis has much improved. The earlier and more intensively the blood therapy is carried out the more brilliant are the results. In cases of melena neonatorum or simple external oozing, the prognosis is fairly good. When the hemorrhage has taken place into the internal organs, the prognosis depends entirely upon the part involved and the extent of the hemorrhage, those involving the lungs and pericardium being very apt to terminate fatally. In cerebral hemorrhage, surgical removal of the clot may serve not only to reduce the high mortality but may also permit a normal development, in cases in which we would otherwise have a paralytic. In cases in which the hemorrhages are minute in character and occur in the brain substance, the infant, should it survive, is most likely to be afflicted with the type of spastic paralysis known as Little's disease, and may also be retarded mentally. The early administration of blood may do much to lessen the extent of this type of hemorrhage, however, and indirectly to promote the early and complete absorption of the clots, thus insuring a normal individual.

OBSTETRIC PARALYSIS

This paralysis is produced by an injury to the nerves of the brachial plexus during birth and is usually of the flaccid type.

The condition was first described in 1768 by Smellie,[1] but it was over 100 years later, in 1872, that Duchenne[1] brought the matter prominently before the medical profession. Erb,[1] in 1874, described the same type of paralysis in adults. Smellie thought the condition was brought about by a long-continued pressure on the arm in the pelvis, while Duchenne recognized that traction on the arm of the child during delivery might be responsible. Erb showed that pressure above the shoulder on the junction of the fifth and sixth cervical nerve roots, the so-called Erb's point, caused the same type of paralysis. Many authors, among whom are Fieux, Schoemaker and Lovett,[2] disagree with Erb, believing that localized pressure on Erb's point would be unlikely. Stransky[1] asserted that the paralysis was probably due to pressure as well as traction, especially if the child was asphyxiated. Kustner advanced a theory that has been rejected by all other writers, that the fracture of bones or the separation of the epiphysis was responsible for the affection. Most authors now agree that the injury is due to overstretching and rupture of the brachial plexus. Frazier and Skillern (1911) resurrect the old theory that the paralysis is caused by the squeezing of the brachial plexus between the clavicle and the first rib, or transverse processes of the cervical vertebræ. Lange, Thomas and others believe that the paralysis is secondary to injury to the shoulder joint. This theory is not upheld by many other authors and is refuted by the work of Sever. Darling[3] and Sharpe[4] have both demonstrated that direct injury of the nerves of the plexus exists in all true cases. Thus it will be seen, that while still some difference of opinion is shown, the more the matter is studied, the more we must be convinced that the condition is nearly always due to overstretching of certain nerves of the brachial plexus.

In Sever's analysis of about 500 cases, 272, or 68 per cent. occurred on the right side. Four hundred were of the upper-arm type. Difficult labor was noted in 418 cases; an anesthetic was used in 363 cases and forceps in 317; asphyxiation of the child occurred in 102 cases, head presentation in 219 and breech presentation in 66 cases; fractures were present in 17 cases and unequal pupils in 16. All his conditions imply the application of force combined with great muscular relaxation of the child, conditions peculiarly favorable to the production of injuries.

Pathology.—There are two well-recognized types of paralysis, the more common of which is that known as the upper-arm type. The other, the so-called lower-arm type, usually is accompanied by some involvement of the upper arm also. The pure upper-arm type results from injury to the fifth and sixth cervical roots, while the lower-arm type includes injury to not only the fifth and sixth cervical roots, but also the seventh and eighth. The lesion usually occurs at the junction of the fifth and sixth cervical nerves. The suprascapular nerve is usually involved in the upper-arm type, and occasionally the first thoracic nerve in the lower-arm type. In mild cases there may be only slight hemorrhage and edema into the nerve sheath, thus possibly causing only temporary paralysis, while in the more severe type actual rupture of the sheath occurs and possibly some of the nerve fibers with resulting scar-tissue formation, and permanent impairment of function. The more extensive the injury, and the scar-tissue formation, the more widespread and hopeless is the paralysis.

Occasionally, the fifth and sixth cervical nerves are completely or partially torn from the spinal cord, and in this case the meninges over the affected area are thickened, fibrous, and adherent to the cord. The affected side of the spinal cord is smaller than normal, and the injured areas of the cord may show evidence of scar-tissue. A reduction in the number of the cells and some degeneration of both the cells and adjacent nerve tracts occur in the anterior horns. Changes have been noted in the cerebrum analogous to those found after amputation of an arm. Boyer reports that the opposite side of the spinal cord may be distorted and otherwise altered by the injury and scar-tissue formation. In most cases the perineural sheath is contracted down on the nerve fibers strangulating them and preventing their regeneration. The nerve sheath repairs itself more rapidly than the nerve fibers thus favoring the strangling of the regenerating nerves. The injury may occur in either vertex or breech presentation, and Sever, in a very carefully conducted series of experiments on the cadaver of the newborn, has cleverly demonstrated the way in which traction causes the various injuries to the brachial plexus with resulting paralysis.

Symptoms.—In the upper-arm type, immediately or a few days after birth, the affected arm lies limp at the side, usually extended and inwardly rotated, with complete inability to abduct, elevate, outwardly rotate or supinate. In this type the deltoid, supraspinatus, infraspinatus, supinator longus, biceps and the teres minor are paralyzed. Occasionally, the supinator brevis, coracobrachialis and the serratus magnus are also involved. As a rule the arm cannot be voluntarily flexed at the elbow, but the flexion and extention of wrist and fingers is not impaired. In this definite and constant paralytic muscle grouping, the injury is located at about the junction of the fifth and sixth cervical nerve roots or Erb's point, just above the point of origin of the suprascapular nerve. It is rather difficult because of the patient's age, to say whether any definite change in sensation takes place. During the first week pain is evident on manipulation, especially in abduction, but this soon disappears. Only very occasionally is swelling or tenderness noticeable over the plexus. The hand grasp is good. The inward rotation persists and as the child grows older some deformity usually develops.

In the lower-arm type we have the same conditions prevailing with the additional paralysis of lower arm and hand, resulting in a useless, dangling arm. Atrophy of the affected parts is seldom noticeable during the newborn period.

Inequality of the pupils may be present and is a most important symptom, as it means that through the injury to the cervical sympathetic nerves definite injury has occurred to the plexus either of the lower cervical cord, the eighth cervical, or the first dorsal nerve, or injury in the spinal cord itself to the fibers of the sympathetic system.

Complications.—Facial paralysis is occasionally seen coincident with and on the same side as obstetric paralysis. It is usually mild and probably due to forceps pressure on the facial nerve. Fracture of the clavicle is not uncommon. Fracture and dislocation of the humerus and separation of the epiphyses have all been noted, but are thought to be merely coincident with rather than the cause of the paralysis.

Prognosis.—The prognosis depends entirely upon the extent of injury to the nerve trunks and cord. In mild cases in which the traction has produced only slight hemorrhage and edema, the entire function may be restored. The prospect for a useful arm in the lower or whole-arm type is very poor. In those cases in which the pupils are unequal, prognosis is especially bad. In the upper-arm type, however, the outlook is very encouraging.

Treatment.—Treatment is limited to an attempt first to prevent the development of deformity, and second, to prevent all possible manipulation of the shoulder during the first few days, thus giving the injuries to the nerves and surrounding tissues every opportunity for rapid repair. The affected arm should be kept warm and the circulation maintained by gentle massage. As soon as the local injury has had time to repair, the involved muscle groups should receive especial attention during the massage. The arm and shoulder joints should be manipulated and exercised, but during periods of rest should be kept in a wire splint or other device in order to remove any drag from the paralyzed muscles, and allow them to regain strength more quickly and prevent deformity. If these measures are carried out properly there is little likelihood that any operative procedure will be necessary or even beneficial in the upper-arm type. The treatment should, however, be continued for several years. Sever does not regard an operation on the plexus in the usual upper-arm type as necessary. In the lower-arm type, the situation is quite different but in any case an operation on the plexus would not be of any great value in restoring functional activity unless contracted and restricting muscles were divided, and careful after-treatment persisted in over a long period.

While many different operators, notably Taylor, Stone, and Fairbanks, have attempted to repair the plexus in the lower-arm type, it is usually found so badly torn, bound down and invaded by scar tissue that any kind of successful repair is very difficult if at all possible, and results in practically no permanent restoration of function. The present most popular method of treatment is to separate the tendons of the contracted muscles, following this as soon as healing takes place, by manipulation, exercises, and massage.

SEPSIS OF THE NEWBORN

Theoretically a discussion of sepsis in the newborn should cover all those diseases caused by microörganisms which result in other than localized symptoms. Clinically, however, the term has come generally to be applied to the more or less general invasion of the human organism by the pyogenic or pus-producing bacteria. The more prominent of these are staphylococcus, streptococcus, pneumococcus, bacillus coli, Friedlander's bacillus, bacillus pyocyaneus, and the gonococcus. Certain microörganisms such as those which produce tuberculosis, malaria, small-pox, syphilis, pertussis, and tetanus produce even in the newborn a definite recognizable symptom complex, and clinically speaking, each disease is a distinct entity, having a characteristic symptomatology and each having its own special pathology and treatment. But, when once the pyogenic group has made a general invasion of the newborn child, there is little difference in the symptom complex produced by them, and from the clinical picture it is very hard to determine in many cases until necropsy, which of these organisms is responsible for the condition. Thus the term "sepsis" in the newborn has come to be used clinically to designate a general infection caused by any of the pyogenic bacteria.

Etiology.—It is well established that the human organism enters the world with a high degree of immunity to certain types of contagious disease, of which scarlet fever and measles are examples. On the other hand, however, we know that the newborn child has little or no defensive power against the invasion of most microörganisms. In other words, the newborn child is a ready host and an easy prey for those microörganisms which ordinarily meet sharp resistance when attacking older children. This lack of resisting power in the newborn is demonstrated by the failure of the regional lymph glands to hypertrophy in the presence of spreading infection, and the low level of the phagocytic activity of the leukocytes, and in many cases, in severe general sepsis, there may be slight or no elevation of temperature. It is not at all easy to understand why the newborn is unable to build up a defence against certain forms of bacteria, as it has been repeatedly demonstrated that he has a high degree of resistance to other forms, except on the theory that the toxic action of certain bacteria is so rapid that the antibodies have not sufficient time for development before the host is overwhelmed.

Extensive research has been made as to the manner in which the infant acquires resistance to certain infections during fetal life but the whole process is still shrouded in a cloud of uncertainty. In the first place, it has been proved by the experiments of Kreidl and Mandl, and also by Wegelius, that the fetal tissues have the ability to react to an invading antigen by the formation of antibodies. Wegelius is quoted by von Reuss as stating that the low or non-susceptibility of the newborn infant for many diseases may be due to an artificial immunization originating from the mother. This may come about, first, by hereditary transmission to the germ plasm of those factors which give immunity to the parents, these factors becoming prominent in the offspring. Or, second, the condition may be due to the same immunizing agent, which exists in the mother, through its action on the germ plasm or on the fetal organism; therefore, an active intra-uterine immunization. Third, antibodies formed in the maternal body may be transmitted to the fetus; thus inducing a passive intra-uterine immunization. Fourth, by the passing of antibodies into the offspring during nursing. Ehrlich, Vaillard, Ehrlich and Hubener, and Wernicke, are quoted by von Reuss as denying that a true inheritance of the immunity of the parents was possible through a transmission by the germ plasm. Pflaundler believes that an active immunization of the fetus in utero is possible only when the specific antigens reach the infant through an imperfect placenta, when the infant itself has been affected with the disease, or when the antigens penetrate at least to the placenta fetalis and incite it to form antibodies. He does not include the transmission of antibodies from the maternal blood to the blood of the fetus as among the physiologic functions of the placenta. von Reuss, however, quotes Wegelius, who worked with the tetanus toxin, vibriolysin, and bacterium coli, as having come to the conclusion that given the presence of antibodies in the serum of a pregnant animal, the antibodies could always be found in the serum of the offspring, and moreover the antibody titration values were higher in the offspring than in the mother animal. From this he concludes that the transmission of antibodies from the mother to the infant is not to be conceived of as a simple process of filtration but that it must be admitted that the placenta possesses a selective power toward the substances present in the maternal serum. Other investigators do not agree entirely with Wegelius's conclusions. To prove the transmission of antibodies by the milk, Ehrlich in his experiment used female mice which had been impregnated at about the same time. One group was immunized with abrin and ricin; the other group was not treated. After the offspring were born he exchanged them, allowing the young ones born of the normal mother to suck the immunized mother, and putting the young of the immunized mother to nurse the normal mother. After some time he found a rapidly decreasing degree of immunity in the latter. In the young of the normal mother, which were, therefore, free from antitoxin, he found an increasing immunity after being nursed by the immunized animal. It is well known that tetanus and diphtheria antitoxin can pass into the milk and many other experiments have shown that the presence of many different types of antibodies in the breast secretion is probable. To be available for the infant in developing immunity, however, such bodies in the milk must be able to penetrate the intestinal wall, and it is not as yet an established fact that such penetration occurs to any large degree.

In general sepsis in the infant, the invasion of bacteria may have occurred before birth, during birth, or subsequent to delivery. The infections which take place in utero may either invade the blood stream of the fetus directly through a diseased placenta or may gain entrance through a contaminated liquor amnii. The normal placenta acts as a line of defense between mother and fetus and it is seldom if ever that the fetus is affected with a disease of the mother while the placenta remains intact. Indeed in many cases in which the placenta itself has been affected, notably in tuberculosis, the child is born free from the disease. However, we know that when the blood stream of the mother carries the organism of certain diseases, such as syphilis and malaria, they are transmitted to the fetus, the invading organism probably passing the placental barrier. Infections taking place through the liquor amnii are probably secondary to the growth of bacteria in the fluid. The liquor amnii may be infected through the intact membranes by way of the vagina, or from the abdominal cavity by way of the fallopian tubes, or, probably most often, infection takes place after a premature rupture of the membranes. The possibility of infecting the infant during its passage through the birth canal is so obvious that it needs no discussion, many pathogenic bacteria being ordinarily present in the vagina. After birth the possibility of septic contamination is of course much greater than previously. The amount of exposure is in direct ratio to the amount of skill and care exhibited in carrying out prophylaxis. Largely because of our better understanding of the necessity for rigid asepsis in the care of the newborn, sepsis is becoming comparatively rare. In the past, especially in lying-in institutions, as high as from 6 to 8 per cent. of all children born would be affected by sepsis in some form or other, while today in institutions in which rigid asepsis and prophylaxis are carefully carried out, the disease is practically unknown. Undoubtedly, the greatest source of contamination of the newborn, has been from the lochia of the puerperal mother, especially in cases in which nurses or others attending the baby, are also in attendance on mothers suffering from puerperal sepsis.

Microörganisms find easy access to the system of the newborn child because of its natural lack of resistance and the unusual number of portals of entry present at this age. The umbilical stump, the digestive tract, the skin, the mucous membranes of the mouth, and the lungs of the newly born offer little resistance to the invasion of bacteria of any marked degree of virulence. While the conjunctiva and the vaginal mucous membranes easily succumb to local infection, they are seldom the source of a general infection. Having entered the system, the infection may become localized in the immediate vicinity of the port of entry, but giving off into the blood stream the poisonous products of its activity, causes a general toxemia. The organism may directly invade the blood stream, causing a true septicemia, or multiple abscesses may develop in the various organs and tissues of the body, a condition usually associated with bacteremia but generally designated as pyemia.

Pathology.—At birth the skin of the newborn is unprotected by a true horny layer and the surface is easily traumatized during birth manipulation and the subsequent cleansing, thus causing abrasions which may readily become infected. Intertrigo, which so easily develops unless great care is taken, is suggested as a possible means by which pyogenic organisms enter the system. Pemphigus, which seldom causes any general symptoms, may destroy the natural resistance of the skin, thus opening the way for more virulent organisms to enter.

The umbilicus is most often contaminated by the careless use of unsterile instruments, ligatures, and dressings, especially by nurses who, caring for puerperal mothers, change the cord dressings without properly cleansing their hands. Likewise, the careless cleansing of the infant's eyes, nose and mouth is often responsible for the infection of these organs. The mucous membranes of the mouth are especially delicate and susceptible to injury from rough cleansing or from unclean nipples which results very often in a stomatitis. According to von Ruess, Czerny and Keller believe that the intestinal wall of the newborn is peculiarly permeable for some bacteria which could not pass through in older sucklings, and that the intestine as a port of entry ranks very high in the production of sepsis neonatorum. They are of the opinion that suppuration of uncomplicated birth fractures is often caused by bacteria that have entered the child's blood from the digestive tract. To the same cause they ascribe the infection of hematomata, and regard many affections of the umbilicus as secondary manifestations of general sepsis. Even icterus neonatorum and melena they believe may be regarded as having some relation to enteral sepsis. Infections of the lungs may be caused by an extension of a rhinitis or pharyngitis, but more often result from the aspiration of contaminated material during or directly after birth. The pathology obtaining in a case of general sepsis depends largely on the port of entry of the organism, the rapidity of the invasion, and, to a lesser extent, on the type and virulence of the bacteria involved. In general there is a degeneration of the tissues of the parenchymatous organs. Meningitis and encephalitis, pneumonia, abscess of the lungs, empyema, peritonitis, otitis media, endocarditis, hemorrhages into the various organs, and purpura are common.

Symptomatology.—Any localized septic affection of the newborn may be the forerunner of a general sepsis. Thus an inflammation of the umbilicus, a stomatitis, a rhinitis, or a skin infection may at any time become systemic. Symptoms depend largely on the severity of the infection. Fever may or may not be present, and when present, may be very high or only slight, continuous or intermittent. Loss in weight, or at best a failure to gain is characteristic. Vomiting is usually present, especially in the later stages. The patient is listless, weak, and usually fails to suckle. The cry becomes plaintive, weak, and distressed. There is a tendency to diarrhea and without proper treatment the patient rapidly becomes dehydrated. The child has a septic appearance; very often there is marked jaundice; hemorrhagic disturbance is frequently found, manifested by a bleeding from the cord, purpura, or as a hemorrhagic pemphigus; the spleen and liver are apt to be enlarged and anemia is profound but, except in those cases in which a positive blood culture is obtained, or the patient is bleeding, there is no marked change in the blood picture, such as one would expect to find in an older child. Nervous manifestations are not unusual, convulsions occurring not infrequently. Chills are never seen in the newborn infant, although there is a marked tendency toward sudden collapse. Meningismus occurs occasionally and general hypertonicity, muscular spasms and tremors are not unusual, although the patient is more apt to be apathetic or stuporous.

Diagnosis.—In all probability there are many cases of mild general infection in young infants which produce very slight if any general symptoms, and are probably recognized only as difficult feeding cases. However, even in well-developed cases of general sepsis the diagnosis may be exceedingly difficult. Fever is a valuable clinical sign when present, but we know it may be absent even in severe cases. Extreme jaundice occurring late in the second week and persistent in character, is very suggestive. Hemorrhagic tendency during the second week is much more likely to be the result of sepsis than to be due to idiopathic hemorrhagic disease. Enlargement of the spleen and liver is of diagnostic value but may be due to other causes. Abnormal quantities of albumin in the urine are rather characteristic. Usually no increase is noted in the number of leukocytes but ordinarily a marked degree of anemia is present.

Prognosis.—In a typical well-developed case of sepsis in the newborn the outlook is very grave, a large percentage of these cases ending fatally. Depending upon the severity of the attack, the disease may last from a few days to several weeks. Undoubtedly, there are many mild cases which recover but it is very unusual in the presence of a bacteremia or a pyemia.

Treatment.—Our greatest asset in combating sepsis in the newborn is a thorough knowledge and an intelligent application of the principles of prophylaxis and asepsis. Infections which occur before or during birth are beyond our power to prevent but proper care *will* prevent the occurrence of infection after birth. Certain principles are fundamental for the proper institutional care of the newborn. First, they should be kept in a separate department or nursery which is especially equipped for their care. Second, nurses caring for newborns should have no care of the puerperal mother, nor should they attend any other case of a septic or infectious nature. Third, nurses attending newborns must themselves be free from respiratory, skin and venereal infection. Fourth, no visitors should be allowed in the nursery or to handle the child while it is being taken to breast. Fifth, mothers with upper respiratory infections should wear a mask while the babe is nursing. Sixth, rigid asepsis must be practised in cord dressings. Seventh, a technique should be carried out by which each babe has its own complete and individual outfit of clothing, sheets, blankets, towels, bath basin, thermometer, etc., these to be kept when not in use in an individual drawer. Eighth, nurses must cleanse their hands after caring for one babe before touching another. If the above technique is properly carried out there is little chance of contamination from other newborns. Once sepsis has occurred we must depend largely upon the maintenance of nutrition and the control of the temperature to combat the disease. There is no condition in which breast milk is so essential as in the case of a newborn suffering from a septic infection. Should the infant be too weak to nurse, breast milk should be obtained and given either by bottle, Breck feeder, or by gavage, depending upon the condition of the infant. It is essential that the body fluids be maintained, as the infant is very apt to become dehydrated if a high temperature persists over any considerable period. This is best accomplished by giving water by mouth between feedings, even by gavage if necessary, by hyopdermoclysis of normal saline to the amount of 50 or 75 c.c. daily, and by normal saline by rectum. A high temperature is best combated by hydrotherapy, and the patient's strength should be conserved by the least possible amount of handling and manipulation. Serum and vaccine therapy so far have been of no value. However, this field is the one of greatest future promise. Subcutaneous injections of whole blood in amounts of from 35 to 50 c.c. have been found of value in combating the anemia and may possibly have some virtue in directly raising the resistance of the patient. In cases in which the anemia is profound and response to subcutaneous injections of blood is indifferent, the introduction of citrated blood into the superior longitudinal sinus to the amount of 30 to 50 c.c. is recommended. When stimulation must be resorted to, hypodermic injections of camphorated oil to the amount of ½ to 1 c.c. will usually give a temporary response. However, for the purpose of sustained stimulation, hypodermic injections of caffein and sodium benzoate in amounts from ¼ to 1 gr. should be given several times daily. Epinephrin in a solution of 1 to 1000 may be given subcutaneously in amounts of from 5 to 15 m. If there is a tendency to collapse, hot baths may be employed if care is used.

FUNCTIONAL DISORDERS OF THE NEWBORN

Shortly after its birth, there becomes apparent in the infant, with more or less regularity, certain disturbances which are not in any sense the result of disease, and may be best designated as "functional disorders." These conditions are, in the normal child, self-limited and cause no material disturbance in its progress. Some of these, such as milk secretion in the breasts of the newborn infant, and vaginal hemorrhage, may be attributed to certain elements entering the blood stream of the child from that of the mother just prior to or at the time of parturition, and they are designated by J. Halban as "pregnancy reactions." Icterus neonatorum, albuminuria, uric-acid infarcts, and inanition or transitory fever are generally conceded to result from inability of the regulating mechanism of the newborn to cope successfully with the demands incident to the metabolic functions of extrauterine existence.

Milk Secretion in the Breast of the Newborn

Von Reusse states that at birth the mammary gland in both males and females forms a little nodule seldom larger than a hemp seed, and that during the first three or four days after birth the gland normally enlarges to about the size of a pea. On the second or third day and always by the end of the fourth day, the mammary gland of the infant, without regard to sex, develops a secretory function, and slight pressure will cause the exudation of a few drops of a clear watery fluid which during the following days will become cloudy and then milky. In early times, this was known as "witches' milk," and was believed by early writers to represent certain pathologic conditions of the gland and to have also some relation to the falling off of the cord. Sinety, in 1885, demonstrated in the mammary gland of the newly born infant, the presence of cul-de-sacs, lined with secreting cells resembling those of the adult, and numerous examinations of the secreted fluid proved beyond doubt its similarity to the early milk secretion of the parturient mother. Genser's analysis is generally accepted as demonstrating the similarity of this secretion in the infant to that of colostrum

Water	95.705
Solid constituents	4.295
Casein	0.557
Albumen	0.490
Fat	1.456
Lactose	0.956
Salts	0.826

Microscopically, the resemblance still persists, milk droplets, leukocytes and colostrum corpuscles being found. Ordinarily, by the eighth day, the secretion reaches its maximum of a few drops and ceases by the end of the second week. In numerous cases, however, the breasts become actually engorged with milk, the glands enlarging to the size of a cherry or even in extreme cases to that of one-half a walnut, and having every appearance in miniature of a functionating adult breast except that the nipples are depressed. In such cases as much as 10 c.c. have been expressed from the breasts with in a few moments. The secretion in such cases may be present for several weeks or even months. As previously mentioned, the theory has been brought forward that this precocious activity in the mammary gland results from the entrance into the infant's blood of certain hormones present in the blood stream of the mother about the time of parturition. Halban asserts that this agent is secreted by the placenta. It has been noted that, in the majority of premature infants, these glands do not function, and the further interesting observation has been made that a parallelism exists between the amount of engorgement of the breasts of the infant and the amount of milk secretion of the mother. No treatment is required in the ordinary case, as the process is physiologic, normal and self-limited.

MASTITIS NEONATORUM

Very occasionally an inflammatory process develops in the mammary gland of the infant. There are several factors which render this possible: The activity of the secretory process; the fact that the milk ducts are open; the demonstrated existence of bacteria in the secretion; and the well-known lack of ability of the newborn to resist infection. Probably the most common cause of mastitis in the newborn is the pernicious habit that ignorant attendants have of squeezing the gland in an effort to express the so-called "witches' milk." Bathing the baby with unclean hands and allowing soiled clothing to come in contact with the breasts are also common sources of infection. The inflammation is usually unilateral, causing a redness and discoloration of the skin, local heat and tenderness and occasionally a slight rise in temperature. There is marked difference in the size of the glands and the process may go on to abscess formation, which may be confined to the breast or may spread into the axilla of the affected side and develop a general sepsis.

The prognosis is ordinarily good, the mastitis responding readily to treatment. As the largest number of cases arise from the indiscreet attempt at expression, caution should be given against it. With the appearance of the first symptoms of inflammation several different remedies may be applied, each with good results. Kerley recommends the application of an ointment containing ichthyol 25 per cent. and oxid of zinc ointment 75 per cent., hot boric acid compresses, or applications of liquor alumini acetatis diluted 8 or 10 times. If abscess develops, prompt incision should be made and free drainage established.

TRANSITORY FEVER

At birth the temperature of the newly born infant is usually that of the mother. Directly after birth the temperature usually falls rapidly from one to two degrees, then during the first few days of life takes on the normal range, as registered in the rectum, of from 97.8 degrees to 99.8 degrees or even 100 degrees. The temperature regulating mechanism is quite unstable and exposure to cold, that would in no way affect the temperature of a month-old child, will cause a sharp drop in the temperature of a newborn. Likewise, an overheated room, or too much artificial heat in the crib will bring about a corresponding rise in the infant's temperature. During the first 10 days it is not uncommon to encounter a slight elevation of temperature, transitory in nature, which often cannot be accounted for by even the most careful examination. This sort of temperature rise, however, bears no direct relation to what we have come to call inanition fever of the newborn. We are not sure, as the name would indicate, just what causes this very characteristic symptom complex. Statistics as to its occurrence differ greatly, no doubt due to the widely different methods employed in the management and care of infants in various institutions. In some instances, the fever has been reported to have occurred in 25 per cent. of 500 consecutive newborns. Various theories have been advanced to explain its occurrence. Clinicians first surmised that the cause was some form of bacterial invasion, but this theory has been generally abandoned because of the extreme regularity of the time of onset and the prompt response to simple methods of treatment. There is also grounds for belief that the rise in temperature may result from the activity of the bacterial flora when milk first enters the intestines. The condition has also been thought to be a starvation or dehydration fever.

This characteristic temperature phenomenon occurs exclusively between the second and fifth or sixth day. For example: A baby, apparently normal in every way, may show on the second or third day a rapid temperature rise to 102 or 103 degrees, followed, after some hours, by a slight remission, but again rising, usually to a somewhat higher point than before, and terminating either by a sharp or a somewhat gradual fall to normal, the whole process occupying from two to four days. The baby as a rule becomes very restless and cries a great deal. The skin and mouth are dry, there is a general hypertonicity, and the pulse and respiration are very rapid. Physical examination discloses no reason for the fever. The stools usually consist only of meconium. However, it is interesting and possibly explanatory in part, that the highest point of fever corresponds in time to the greatest loss of weight and that the temperature immediately subsides with the first gain. Also to give food and water in generous quantities will promptly terminate the fever. The explanation would be simple if this temperature rise occurred only in debilitated, undernourished or dehydrated infants, but the facts are that the apparently well-nourished, strong, vigorous newborn develops this disorder as readily as another. There is nothing contagious or infectious in the condition since seldom is more than one infant affected even in a nursery which may contain 20 others, being cared for exactly alike. It is interesting that infants developing this fever are always obtaining a very small amount of breast milk, but, on the other hand, many other babies in the same state of nutrition obtain just as little without developing the fever. The experience of the writer rather bears out the assertion that starvation plays some role in its production. Three years ago the newborns at Hillcrest Hospital were on the obstetric service and no particular attention was paid to the amount of milk obtained during the first few days of life. Under this arrangement, typical cases of transitory fever were of not infrequent occurrence. After the newborns were put on pediatric service, a rule was established that every child who did not obtain 2½ oz. of milk at breast after the first 24 hours, was to be given expressed breast milk or milk mixture to complement that amount. Since the establishment of this rule not one typical case of transitory or inanition fever has occurred. A few infants have shown a sharp temperature rise on the second day, but this has subsided within 24 hours with no other treatment than a little additional water. Whatever the cause may be, the treatment is simple and effective: Cool packs, forced feeding up to at least 2 oz. every four hours with 2 oz. of water in the intervals, the water and milk to be given by tube if necessary. Vomiting seldom occurs in these cases, but hypodermoclysis of normal saline may be resorted to if necessary.

FACIAL PARALYSIS OF THE NEWBORN

Facial paralysis of the newborn almost always results from injury to the peripheral trunk of the facial nerve. It may be, however, the result of some intracranial pathology. For practical diagnosis and treatment, therefore, facial paralysis may be broadly divided into two classes, namely, those of intracranial, and those of extracranial origin.

Injuries of the extracranial variety are practically always due to some form of traumatism to the main trunk of the facial nerve near its exit from the stylomastoid foramen or at the point of crossing the ramus of the inferior maxilla. This form of injury, with few exceptions, results from trauma, the blade of the forceps having been so applied to the head as to press directly upon this area. Facial paralysis of this type is, therefore, usually unilateral. The injury is said to occur occasionally after spontaneous delivery, but ordinarily only when the pelvis is so flat or contracted as to cause extremely prolonged pressure over the trunk of the facial nerve by the promontory or the symphysis. Cases have also been cited in which a hematoma in the upper portion of the sterno-cleido-mastoid has involved the facial nerve, and other instances have been noted in which pressure by the shoulder has been sufficient to cause paralysis. The degree of injury to the nerve trunk can be judged to some extent by the amount of superficial swelling and contusion, but not with great certainty as the nerve trunk may be merely involved in a surrounding hematoma or edema, or there may be an actual stretching of the nerve trunk. In the peripheral type of injury both lower and upper portions of the face are paralyzed, giving a smooth, rounded, swollen appearance to the involved side, with the mouth drawn to the unaffected side when the infant cries, and an inability to close the eye on the paralyzed side. The paralysis usually becomes apparent on the first or second day and may have disappeared entirely by the fifth or sixth. Ordinarily, recovery is complete by the end of the second week, but very rarely it may persist for a week or two longer and occasionally may leave some permanent atrophy. As a rule, the paralysis does not affect the normal progress of the child, and, except for the grotesque appearance, would attract no attention. Suckling, which is largely carried on by action of the tongue, is not interfered with.

No treatment is required in most cases, other than to protect the open eye and keep it thoroughly cleansed and moistened. Electrical stimulation to the paralyzed muscles is advised in cases in which recovery has not begun by the end of the second week.

Facial paralysis of the second group usually result from internal hematoma from intracranial hemorrhage, may be either unilateral or bilateral, and seldom involve the muscles of the upper half of the face. Facial paralysis from this cause is usually accompanied by paralysis involving other portions of the body and by the general symptoms of intracranial hemorrhage. As the treatment of these two groups is so different, the differential diagnosis should be established as early as possible. In the first type, the well-being and general progress of the infant is unaffected; while in the second a small circumscribed hemorrhage directly over the cortical center of the facial nerve would produce a localized facial paralysis. This condition rarely occurs, and when it does is accompanied by general symptoms of cortical irritation. Also in intracranial injury, the centers of the hyperglossal nerve are very likely to be affected, causing a paralysis of the tongue. On investigation, the tongue, as it lies in the mouth, will be seen to be twisted toward the affected side. The tongue is rarely affected in paralysis of the peripheral type. Another point of differential diagnosis is that, in the intracranial type, the upper half of the face is not ordinarily involved because the nucleus of the seventh nerve supplying that area derives some of its fibers from both sides of the brain, while the part of the nucleus supplying the lower half of the face derives its fibers entirely from the opposite cortex. It is often difficult to determine in the newborn whether or not the upper face is involved. Occasionally, facial paralysis of the intracranial class may be of congenital origin due to degenerative processes involving the cortical centers of the seventh nerve.

The prognosis and treatment in these cases will depend upon the location and extent of the hemorrhage, as outlined in the chapter on intracranial hemorrhage.

BONE INJURIES IN THE NEWBORN

Aside from injuries to the skull one may encounter fractures and dislocations affecting the skeleton of the trunk and extremities. Most of these injuries are directly the result of artificial delivery, and the less skillful the operator, the more likely are they to occur. Injury to the vertebral column practically never occurs except in breech extractions. The spine is very elastic and owing to its partly cartilaginous formation, has great flexibility and will endure a very considerable amount of torsion and strain without fracture or dislocation. In fact, there is much greater likelihood of injury to the cord and surrounding meninges than to the column itself. Lesions of the lower cervical vertebræ are the most frequent and consist chiefly, according to Stolzenburg, in tearing off the capsule of one of the lateral joints, or in the rupture of neighboring parts of the intracranial ligament and rupture of the vertebral body through a plane of cartilage. As a rule, severe injuries to the vertebral column result in such injury to the cord and meninges as to be incompatible with life. According to von Reusse, Birnbaum describes the symptoms as follows: Breathing either fails to begin at all, although the heart functions for a time, or breathing ceases immediately, before any disturbances of the heart's function appear. It may happen that hemorrhage is at first checked, but is started again by movement. Although the life is usually very brief, it may endure for some days.

Fracture or dislocation of the ribs almost never occurs, and when it does can only result from unnecessary violence in gripping the chest by the operator either in delivery or in an effort at resuscitation.

Fracture and dislocation of the bones of the shoulder girdle are occasionally encountered. Fracture of the scapula rarely occurs, but separation of the epiphyses is not unusual. The clavicle, however, is more commonly fractured during delivery than any other bone of the body, some statistics placing the frequency at 1 per cent. of all births. This fracture usually results from manipulation during artificial delivery, but is by no means rare after spontaneous birth, especially in multipara, due no doubt to the intensity of the labor pains. The injury is commonly in the middle or outer third and there is seldom much deformity. Nothing may attract attention to the fracture, as the infant uses the arm on the affected side without apparent discomfort. Just at first crepitation is easily obtained and there may be slight swelling over the point of fracture. By the end of the second week callus formation usually becomes prominent enough to be noticed. Even without treatment, fracture of the clavicle tends to heal rapidly in good position, and although there should be slight deformity, the bone gradually assumes its normal curvature with the development of the trunk.

The only fractures and dislocations of the extremities which occur in any appreciable number are fracture of the middle or upper third of the femur, or middle or upper third of the humerus, or very uncommonly, dislocation of the shoulder joint. Fractures of the forearm and lower leg as well as dislocation of the hip, while they do take place, are so infrequent as to be looked upon as curiosities. Fractures of the femur or humerus result, in most cases, from hooking the fingers around these bones and exerting extreme traction in an attempt to bring down the arm or leg. Separation of the epiphyses in the newborn is caused from the same force which, in an older child, would result in dislocation of the shoulder. In fracture of the long bones of the newly born child, to a somewhat lessened degree, the same deformities, crepitations, tenderness, and pain are found that we encounter in similar injuries in older children and adults, but the tendency is toward extraordinarily rapid union without deformity, largely owing to lack of muscle spasm.

The Roentgen-ray is invaluable in the diagnosis of fractures of the long bones, but is of little assistance in the study of dislocations or separation of the epiphyses, because of the softness of the bony structure in the joints.

In the treatment of fractures of the newborn, the same rules of immobilization, correction, and prevention of deformity apply as in their treatment in older children and adults, bearing in mind, however, that the tendency to rapid union without deformity obviates the necessity of prolonged application of heavy splints or appliances which may do serious injury to the tender skin or soft parts beneath.

CAPUT SUCCEDANEUM

These tumors occur as an infiltration of the connective tissue on the presenting part of the fetus, and are not uncommonly seen in the newborn.

At birth the soft parts of the birth canal develop such pressure and constriction upon the presenting part of the fetus as to produce a stagnation of the circulation of blood and lymph, resulting in a serosanguineous edema of the connective tissue of that part.

This condition occurs most often in head presentations, there appearing, usually over the vertex in the upper occipital or posterior parietal region, an edematous tumor of irregular outline, ranging in size from a slight swelling to one involving a third of the scalp surface. Usually the size is dependent upon the duration and severity of the labor. These swellings rapidly disappear, absorption as a rule being complete by the end of the fourth day, and no treatment is required.

Caput succedaneum is sometimes confused with cephalhematoma, which occurs in the same region and at about the same time. Differentiation is not difficult, however, in asmuch as the cephalhematoma is a fluctuating mass, the boundaries of which are always limited by the sutures of the cranial bones, while the caput succedaneum pits on pressure, and may, and usually does, extend across the sutures.

In the case of face presentation, similar edematous swellings of the face occur, or, in breech presentation, they may appear on the external genitals.

HEMATOMA AND MYOSITIS OF THE STERNOCLEIDOMASTOID MUSCLE

Hematoma which develops in the body of the sternocleidomastoid muscle occurs rather rarely and may appear during the first few days after birth or as late as the second or third week. It may attain the size of a pigeon's egg, and on palpation gives one the impression of being of a hard cartilaginous consistency. It is movable with the muscle, and although not painful is often tender to the touch. The skin covering the tumor is not discolored and may or may not be edematous. It occurs much more frequently on the right than on the left side, but in rare instances both sides may be involved. The head is often drawn toward the affected side with the chin rotated away from it, but in some instances the normal position of the head is not affected.

The condition results from a laceration of the blood-vessels and fibers of the sternocleidomastoid muscle with the consequent infiltration of blood, accompanied by inflammation of the muscle and its sheath. It occurs when there is extreme rotation of the head during parturition, and is not an evidence of the improper use of forceps in delivery, as it more commonly occurs in unassisted labor. The percentage of cases is greater in breech than in vertex presentations.

These tumors slowly diminish in size without treatment and ordinarily disappear during the second or third month of life. The assertion that permanent wry-neck is sometimes the result of hematoma of the sternocleidomastoid seems to be without proof.

Brief and gentle massage of the muscle may be employed each day or a moderate rotating of the head to forestall any stiffening of the neck, but any further treatment is unnecessary, and operative interference is never indicated.

CEPHALHEMATOMA

These tumors of the head are occasioned by ruptures in the vessels of the subperiosteal zone with the resulting accumulation of blood under the periosteum. The swellings vary in size from that of a marble to that of a goose egg, usually attaining their maximum about the end of the first week of life. Their occurrence is decidedly more frequent in boy than in girl infants, almost two to one, possibly because of the larger size of the male head. According to the best statistics, they are rather rarely seen, in from four-tenths to six-tenths of 1 per cent. of all births.

Cephalhematomas are in all probability produced during parturition. As the head is moved forward and back by the uterine contractions, the covering is worked loose from the bones by the pressure of the soft parts of the birth canal, and the ruptured vessels of the subperiostial region pour out the blood which forms the tumor. In cases of bilateral hematoma, the injury may often be traced to pressure from the forceps. These tumors are more often observed in the infants of primiparæ, especially elderly mothers, than in multiparæ, because of the rigidity of the birth canal. They are also more common in full term than in premature infants. If the cephalhematoma is growing rapidly in size, the possibility of a coincident hemorrhagic disease of the newborn must be considered, and should the bleeding and coagulation time be unduly delayed, the routine treatment as indicated under hemorrhagic disease should at once be instituted.

These tumors are most often seen over the parietal bones, the right side being the one more often affected, as it is the surface most often presenting, but they occasionally occur over both parietals, or over the frontal, occipital or temporal bones. The skin covering the tumor is not discolored, but during the first days after birth is apt to be edematous, giving the swelling a doughy consistency which gradually changes to one of distinct fluctuation.

The hemorrhage disturbs and lifts a portion of the osteogenetic zone, as well as the periosteum, resulting in a characteristic symptom of cephalhematoma—the formation during the second and third week of a ring of newly formed bony tissue at the periphery of the tumor, which grows toward the center until sometimes a part and sometimes the entire surface is covered by this newly formed parchment-like bone, giving out a distinct crepitation on pressure. In isolated cases, an internal cephalhematoma may form on the inside of the skull, the hemorrhage having passed through a fracture or fissure in the skull and spread between the bone and the dura mater. These tumors are usually of small extent and diagnosis can only be made by cerebral symptoms.

The absorption of the effusion takes place gradually and requires from four to six or eight weeks or even longer, according to the size of the tumor. As a rule, no trace of the swelling will be observable after its retrogression except occasionally a slight periosteal thickening or roughness of the bone.

The most characteristic diagnostic feature of cephalhematoma is that, owing to the extravasation taking place between the periosteum and the cranium, the boundaries of the tumor never cross the sutures of the bones, the periosteum being much more firmly adherent at the sutures than over the surface of the bones. The stability and position of the hematoma differentiates it from the meningocele which may be reduced by pressure and which fluctuates with respiration. During the first day or two after birth the cephalhematoma may be hidden under a caput succedaneum, but with the disappearance of the edema the characteristic fluctuation of the hematoma will appear.

Suppuration is the one condition to be guarded against, therefore the head should be protected from the slightest injury. This constitutes all the treatment usually necessary. Only in very large hematoma that fail to absorb in a reasonable time, or in case of suppuration, should an incision be considered, and then only under the strictest asepsis. The prognosis in an uncomplicated cephalhematoma is good.

SKULL AND BRAIN INJURIES

Injuries to the Skull

In fully 45 per cent. of newborns, to a greater or less degree, a bending or molding of the cranial bones will be found, due to the passage of the head through the pelvic canal. In some instances, this is accompanied by overriding of the bones at the sutures. Molding and overriding may occur to an extreme degree without apparent discomfort or injury to the infant and are frequently accompanied by caput succedaneum and cephalhematoma. The overriding ordinarily subsides within the first three or four days, and the molding, depending upon the degree, may disappear by the end of the second day or persist to some degree for many months.

Occasionally permanent depressions are produced most often in the parietal or frontal bones, usually by pressure of the promontory, especially if the anteroposterior diameter of the pelvis is shortened. These depressions are generally recognized as either spoon shaped or grooved and ordinarily do not produce symptoms. Occasionally in the deep-grooved form there is a fracture of the inner table of the skull which may be the seat of hemorrhage. Many times these depressions are very slight and probably disappear immediately without recognition, as there is no superficial contusion or abrasion of the scalp. Even in rather large depressions of the spoon-shaped variety, a spontaneous return to normal occurs. In the deep-grooved type, however, the condition is apt to persist unless there is operative interference. Many of the depressions can be reduced by simply holding the head in the hands and making generalized pressure toward the seat of the depression. When actual operative interference is necessary, various methods have been used, either trephining and introducing a flat instrument through the opening to raise the depression, or by reaching it through an opening made into the nearest suture. However, either of these procedures may possibly occasion serious cranial injury and should not be resorted to without grave consideration. The method of choice is that used by Vicarelli whose instrument is designed to screw into the depression and elevate it from the outside.

Fractures of the skull bones are comparatively infrequent. Occasionally in severe forceps manipulation, but rarely in spontaneous delivery, fractures of the parietal or frontal bones are produced. This is likely to occur only when the skull bones are abnormally thin and the pressure exerted is great. The fractures may be single or multiple and, according to Hartmann, usually take the direction of the trabeculæ of ossification which run radially from the frontal and parietal eminences toward the periphery of the bone. Many times these fractures occur without causing any serious injury to the brain or meninges, but are often accompanied by external and possibly internal cephalhematoma. However, conditions so severe as to bring about injuries sufficient to cause fracture of the cranial bones may well produce serious injuries to the meninges and other soft parts.

Rarely a so-called traumatic meningocele or cephalic hydrocele may develop through the open fracture. Occasionally fractures at the base of the skull are brought about by extreme traction exerted in breech delivery on the after-coming head, or by forceps applied in vertex presentation, the usual injury being fracture and overriding at the juncture of the condylar and supraoccipital portions of the occipital bone. This injury is usually fatal because of the hemorrhage involved, and, according to von Reusse, because of the narrowing of the foramen magnum which always takes place.

INTRACRANIAL HEMORRHAGE

No greater menace jeopardizes the life and future well being of the newly born child than intracranial hemorrhage, with its trail of suffering, idiocy, epilepsy, paralysis and death. Cerebral hemorrhage has been reported present in as high as 50 per cent. and in one series, 76 per cent. of deaths of the newborn, and thus is an important factor in more than one-half of the deaths occurring during the first days of life. As will be noted later in the discussion, many cases of intracranial hemorrhage occur in which the infant survives without subsequent deleterious effect, while many others become mentally defective, more or less paralyzed or possibly epileptic.

Intracranial hemorrhage has until very recently been supposed to be due entirely to extreme congestion, injury and rupture of the intracranial blood-vessels from force or pressure to the head incident to birth, and this undoubtedly is the cause of the cerebral bleeding in a large percentage of cases. Recent investigations, however, lead us to believe that in many instances the hemorrhage results from a general condition known as hemorrhagic disease of the newborn, and that, therefore, in many cases of extensive intracranial bleeding, direct traumatic injury to the cerebral blood-vessels has not occurred. This type of cerebral hemorrhage has been fully considered in the chapter on hemorrhagic disease, the discussion here being confined to traumatic hemorrhage.

Unusually hard, difficult and prolonged labor is generally considered as amplifying the danger of cerebral hemorrhage, especially when instrumental delivery has been necessary. Distortion of the skull, overlapping of the cranial bones resulting in laceration or rupture of one of the cerebral veins or capillary vessels, is most frequently the source of the bleeding. Occasionally the hemorrhage may be caused by the tearing of the veins opening into the superior longitudinal sinus. It has been suggested also that veins entering the transverse sinus may be similarly injured. In by far the largest number of cases of cerebral hemorrhage, however, the exact bleeding point can not be determined, owing to the general congestion of the cerebral blood-vessels.

In a general way hemorrhages are more common posteriorly and at the base of the brain than over the convexity or in the frontal region. Hemorrhages outside of the dura are seldom found and when present are usually internal cephalhematoma and result from fracture. The hemorrhages are customarily situated, according to Kundrat, under the arachnoid and in the tissues of the pia. Symptomatically Seitz classifies hemorrhages into four types, namely: (1) Supratentorial; (2) infratentorial; (3) combined; (4) lateral ventricle. In the supratentorial type, the bleeding occurs from injury to the veins entering the superior longitudinal sinus or from the margin of the tentorium, the blood usually flowing over the cerebellum. The hemorrhage as a rule is unilateral. In the infratentorial type the bleeding is from the region of the transverse sinus or the margin of the tentorium and flows over the cerebellum around the cerebral peduncles and the medulla oblongata and even into the spinal canal. In the fourth type the blood enters the lateral ventricles from bleeding in the region of the straight sinus, of the inferior longitudinal sinus and of the veins of Galen.

In pure cases of traumatic hemorrhage, symptoms are present at birth or appear very early. The later the symptoms appear the less likely the hemorrhage is to be of traumatic origin. The symptoms are essentially those arising from increased intracranial pressure or cortical irritation and, therefore, many times, small hemorrhages may be present without giving rise to symptoms. When the hemorrhage is massive at birth, the infant is often still born or is made to breathe only after prolonged efforts at resuscitation. This is especially true in large extravasations of the infratentorial type. Should the resuscitation be successful the child may apparently do well for a few hours or may immediately show signs of the increased cranial tension, by restlessness, refusal to nurse, wakefulness, a peculiarly distressing cry suggestive of pain, a strong full pulse, sometimes increased pressure in the fontanelle, probably pallor, increased reflexes, at first twitchings and later convulsions, followed by stupor and often by death from respiratory failure. Convulsions are probably the most constant and characteristic symptom of cerebral hemorrhage. Severe hemorrhage of the infratentorial type may be present without convulsions, death occurring rapidly through interference with respiration and the heart centers. In the supratentorial type the hemorrhage is often unilateral and may produce merely localized twitchings of the face or eyes, or of one extremity, or may involve one complete side, thus pointing out the probable seat of the hemorrhage. It is more likely, however, that the early localized twitchings will be followed by generalized convulsions. Should the infant survive the acute disturbance immediately consequent upon the actual bleeding, the extent of the permanent injury will depend largely upon the location and size of the clot as well as the amount of absorption which takes place. In the milder cases, undoubtedly the clot may entirely absorb without permanent injury, while in other cases where apparent recovery has taken place, muscular weakness and possibly mental retardation will become noticeable after the sixth month. Particularly in this type much unexplained crying occurs during the early months. In the more severe cases in which the child survives the initial disturbance, we have the spastic paralysis and ofttimes marked mental deficiency, or in other cases recovery may be followed by epilepsy and sometimes by hydrocephalus. Minute hemorrhages into the brain substance are often coincident with meningeal hemorrhage, but may occur independently and have even been noted as occurring antepartum. It is not unlikely that hemorrhagic disease plays a more prominent part in hemorrhages of this type than does traumatism.

Little progress has been made in the treatment of hemorrhage of the traumatic type, but as may be seen by referring to the chapter on hemorrhagic disease, many cases of intracranial hemorrhage may be prevented and many cured by attention to the coagulation and bleeding time, which should be routinely tested on the second and fourth day in a newly born infant. After an especially difficult labor or when there is anything abnormal in the progress of the infant, the bleeding time should be tested as often as twice daily. The treatment of cerebral hemorrhage must be largely symptomatic. The patient should be kept warm and quiet, and cared for with the least amount of handling possible. He should not be put to breast until all signs of irritation have subsided. When convulsive twitchings appear, sufficient chloral hydrate should be administered by rectum to control the convulsions—usually from 3 to 5 gr., repeated when necessary, will be found sufficient. Cold compresses applied to the head may be of some benefit. Repeated spinal puncture, which has been occasionally recommended, is only justified when extreme bulging and tension of the fontanelles appears, as the manipulation and struggling incident to spinal puncture will do more harm than the procedure will do good. When the hemorrhage has been definitely located in an accessible area and the coagulating and bleeding time have been normal, surgical removal of the clot by a trephining operation has been successful in a number of instances. In the general care of hemorrhagic cases careful attention to the feeding must be maintained. As before stated, the infant should not be taken to breast, but should be fed expressed breast milk in small amounts at frequent intervals. If the baby is unable to suckle, it may be fed with a medicine dropper, but if it does not swallow readily it should be fed by the use of a tube. Dehydration must be prevented by giving saline enemas or by hypodermoclysis when sufficient fluid cannot be administered by the mouth.

EDEMA NEONATORUM

Edema neonatorum is the term used to indicate a condition outwardly resembling somewhat sclerema neonatorum but characterized by an infiltration of a serous fluid into the subcutaneous tissues.

Etiology.—The onset occurs as a rule during the first few days of life, and the causes may be varied, but ordinarily the disease is seen in the feeble, ill-nourished or premature infant, and frequently is secondary to some cardiac weakness. Among the other primary sources suggested are atelectasis, nephritis, hereditary syphilis, erysipelas, and undue exposure to cold directly after birth. Necropsy usually discloses some lesion of the internal organs or vascular system, but cases have been reported in which no definite pathology could be found. It is suggested that cases of this type may be mild sclerema rather than edema.

Pathology.—As indicated under etiology, most writers attribute true edema neonatorum to a disturbance of the cardiac, pulmonary, renal or vascular system, the condition in the child being similar to general anasarca in the adult except that effusion into the serous cavities is rare.

Symptoms.—The edema, commonly occurring before the third day, is noted first in the dependent parts, and the regions most affected are those of loose cellular structure, such as the eye lids and the scrotum. The first signs of swelling are frequently noted in the lower part of the legs or in the face. When the edema appears on the lower limbs, it gradually spreads upward; the hands are next involved, then irregular distribution follows over the face and trunk. The affected parts appear swollen and have a doughy consistency to the touch. There is marked pitting on pressure and movement of the joints is slightly restricted due to the edema in the surrounding tissues. The infant is generally somnolent and it is difficult to arouse him sufficiently to nurse. The temperature is subnormal, the circulation feeble, the respiration slow and superficial. Urinary findings depend upon the amount of renal involvement. In color the skin is usually livid, but may be jaundiced from accompanying icterus.

Diagnosis.—The disease most likely to be confounded with edema neonatorum is sclerema. The chief differences are in the distribution, the pitting on pressure in edema, and the absence of that dry, leathery feeling of the skin so characteristic of sclerema, and which is usually accompanied by a general rigidity.

Prognosis.—The prognosis is commonly grave, with a mortality of 80 per cent., death usually occurring before the tenth day, but the outcome depends largely, of course, on the underlying cause and the degree of involvement. Many cases have been reported in which spontaneous recovery has taken place.

Treatment.—A general effort is made to conserve the body heat and to maintain proper nourishment.

WINCKEL'S DISEASE

Winckel's Disease is a very rare affection of the newborn, characterized by a profound icterus, cyanosis, and hemoglobinuria. It usually occurs epidemically in institutions. The progress of the disease is rapid with a very high mortality. In 1879 Winckel described the symptom complex now known by his name as a hitherto unknown disease of the newborn. His report covered 23 cases occurring epidemically in the same institution, the disease affecting one-fourth of all children born during the period of its occurrence. Winckel, however, was mistaken in his contention that he was describing a previously unrecognized entity, as Charrin had described the disease in 13 cases in a report published in 1873. M. J. Parrot published two cases in the same year and a case of this disease had been the subject of a thesis by Biglow in 1875. Wolczynaki reported two epidemics of similar nature in 1893. Sporadic cases have been reported by others from time to time, Brandenburg having been able to collect 50 cases from the literature up to 1911.

Etiology.—The epidemic character of the disease points unquestionably to some infectious or toxic origin. Numerous bacteria have been found in the organs at necropsy such as bacillus coli, streptococcus, and pseudodiphtheria bacillus. There is no distinct history of a constant umbilical infection, so that the umbilicus is not thought to be the portal of entry. Winckel described his cases as being due to an infection of the gastro-intestinal tract thought to be caused by the administration of polluted water. In nearly all reports, however, the type and source of infection or poisoning remains very obscure.

Pathology.—Those cases which have been studied pathologically have uniformly shown changes in the kidney. As described by Bass the cortex is swollen, dark, and subscapular extravasations of blood may occur. The pyramids are the seats of hemoglobin infarcts which appear as dark striations. In the tubules of the kidneys hemoglobin crystals are rather characteristic. Small hemorrhages are noted in the gastro-intestinal mucous membrane. Peyer's patches are swollen and the mesenteric lymph-nodes are greatly enlarged. The spleen is swollen and hard and filled with blood pigment. Small hemorrhages are found throughout most of the other viscera.

Symptomatology.—The disease usually appears about the fourth day but may be seen as late as the tenth or twelfth day. The temperature usually remains undisturbed although in one or two cases reported a temperature of from 101 to 103 degrees has been noted. The ordinary icterus becomes rapidly more intense until it assumes the deep bronze color of the mulatto, the discoloration being the result of the deposit of blood pigment in the skin. Often the sudden onset of a persistent cyanosis is striking. The urine is frequently passed, contains large amounts of hemoglobin and has a dark-brown smoky appearance. The general restlessness, prostration, increased pulse and respiratory rate are the same as would be expected in general sepsis. In most cases the spleen has been reported as large and very hard. An examination of the blood shows a large increase in leukocytes, the appearance of many granules, a low red-cell count with a large number of partially broken down red cells. At the point of puncture the blood is chocolate brown in color as it exudes from the skin.

Prognosis.—The disease is usually fulminating in character, death having occurred as early as nine hours after the first appearance of symptoms. Two or three days marks the usual run of the disease in the fatal cases but death may occur as late as at the end of two weeks. The mortality is very high, having amounted to 82 per cent. in Winckel's cases.

Treatment.—The malignancy of this infection is so great that treatment is of little avail. Because of the epidemic character of the disease every precaution to carry out strict isolation should be instituted. General supportive measures as suggested in sepsis are of value.

ATELECTASIS

By congenital atelectasis is meant a failure of the whole or any considerable portion of the lungs of the newborn to expand after birth.

Normally a day or possibly two will pass before all parts of the newborn's lungs are functioning. Von Reuss states that at the end of the first 20 minutes the air capacity of the lungs is 17 c.c., at the end of three and one-half hours it has reached 21 c.c., and at six hours it is approximately 36 c.c. The volume increases rapidly during the first day only, and it is days and sometimes weeks before atelectasis has entirely disappeared. Many times in infants several weeks of age, who have died from some unallied cause and have shown no signs of cyanosis or respiratory distress, post-mortem examination has shown as much as two-thirds of the lungs to be in a state of atelectasis. It is said that a greater degree of atelectasis is usually found in the left lung than in the right. Also the posterior part of the lower lobes, the paravertebral and those portions around the hylus are the last to expand, and it is in these portions that the atelectasis is the most persistent.

Etiology.—Atelectasis is apt to persist in children who have been born asphyxiated, in prematures, cases of cerebral hemorrhage, or edema of the brain from severe pressure, and in children who are of low vitality. Failure of the proper functioning of the respiratory center is held responsible for the lack of lung expansion, except in cases in which there is compression of the lungs from pleural effusion, constriction of the lumen of the trachea from congenital anomalies, or an enlarged thyroid or thymus; or a plugging of the bronchus. The failure of the respiratory center is attributed to the same causes in atelectasis as in asphyxia, the two conditions being usually closely associated. In the case of prematures and other feeble infants, it has been suggested that the lack of muscular activity is responsible for a diminished carbon dioxid content in the blood, and is thus indirectly the factor leading to the weak action of the respiratory center.

Pathology.—Children dying during the first few minutes after birth, will show almost complete atelectasis of both lungs, and in those who have survived even for several days the expanded portions may still be very small. In these cases the lungs show little pathological change except the atelectasis. In those parts in which this condition is complete, there is little change from the fetal state and the sections may be readily inflated. They are dark brownish red in color, do not crepitate, and will not float. When the atelectasis has been of long standing the parts affected are very likely to be the site of infection, areas of pneumonia and congestion, with occasional hemorrhages. When the atelectasis is of the acquired type, as when it is due to the plugging of a bronchus, the process will be more disseminated. In nearly all cases in which breathing has been going on for some days, areas of emphysema will be found in the expanded portions.

Symptoms and Diagnosis.—Cyanosis otherwise unexplained in the newborn is always suggestive of atelectasis. The symptom complex depends largely on the vitality of the infant, the degree of atelectasis, and the associated conditions. In those cases of congenital asphyxia in which resuscitation has been difficult, and atelectasis remains to a marked degree, the infant will lie relaxed and more or less somnolent, breathing very superficially and at times almost imperceptibly, developing sudden attacks of cyanosis, with possible convulsions. This condition usually terminates fatally. In more vigorous infants in whom asphyxia is playing a lesser role and there is a large degree of atelectasis, breathing will be rapid and more violent in character, there will be less permanent cyanosis and the respiratory effort will often be accompanied by lateral retraction of the chest or marked depression of the lower sternum. In this type of case if the atelectasis is not due to obstruction, the prognosis is relatively good. In the other type, so often found in prematures and weaklings, the respiratory excursion will be slight, and there may be no other clinical evidence of atelectasis except transitory attacks of cyanosis. It has been suggested by Holt that many infants who show signs only of feeble vitality and malnutrition are primarily cases of partial atelectasis. It is this type of case which is most likely to succumb to infection.

Physical signs are of little aid in arriving at a diagnosis of atelectasis. Percussion gives us little information except in cases in which marked difference shows in the resonance of the two sides of the chest. The usual signs of consolidation, such as bronchial breathing, cannot be heard but slightly diminished breath sounds may be detected over the areas of atelectasis. The most reliable sign is that described by Peiser, known as atelectatic crepitation which is often obtained over the consolidated portions on deep inspiration. An *x*-ray examination may also be of value.

Treatment.—In the early stage, treatment is the same as that outlined for congenital asphyxia with which it is usually associated. After the initial resuscitation, the infant suffering from atelectasis should be made to cry vigorously once or twice daily. This is best accomplished by cutaneous irritation, as spanking, cold spray, etc. It is also important that these babes should not be allowed to be in one position for any long period. They should be turned first on one side and then the other from hour to hour. For the cyanotic attacks, external stimulation and hypodermic injections of camphorated oil and sodium benzoate are recommended.

ASPHYXIA NEONATORUM

(Apparent Death)

Asphyxia neonatorum is a grave condition of lowered vitality which more or less simulates death and develops during or immediately following birth. It is due to the suspension of vital phenomena, resulting from interference with the proper oxygen and carbon dioxid interchange in the blood of the infant.

Asphyxia is usually divided into two classes: First, intra-uterine or congenital asphyxia; second, post partum, or acquired asphyxia. The first is the condition usually referred to when the term asphyxia is used, and this class is further divided into asphyxia livida, or first degree; and asphyxia pallida, sometimes spoken of as second degree. Class two or acquired asphyxia is closely related clinically and pathologically to the condition usually described under the term atelectasis.

Etiology.—Any condition during labor which, for any appreciable length of time, interferes with the normal circulation of the blood through the normally functioning placenta, at a time when the infant lungs are for any reason unable to commence natural respiration, will bring about a state of asphyxia in the child, the degree of the asphyxia depending upon the length of time and amount of the interference. The things which are usually designated as being responsible for fetal asphyxia are: Tetanus uteri, commonly caused by unwise administration of drugs to stimulate uterine contractions; prolonged delivery; tearing, twisting or compression of the umbilical cord; premature detachment of the placenta, eclampsia or pneumonia in the mother; failure of the maternal circulation for any reason; and abnormal cerebral pressure in the fetus. Ordinarily in the course of normal labor the placental respiration continues until the child is delivered and through this agency the normal exchange of oxygen and carbon dioxid takes place in the infant's blood until the moment of birth, at which time the respiratory center in the medulla of the infant is stimulated to action, reflexly through irritation of the air or other foreign substance on the skin, and by an excess of carbon dioxid or a deficiency of oxygen in the blood stream, due to the discontinuance of the placental respiration. In the development of fetal asphyxia interference with placental circulation from any of the previously mentioned causes and the consequent disturbance of the carbon dioxid-oxygen balance in the blood stream is supposed to incite the premature stimulation of the respiratory center in the infant, causing attempts to breathe before the head is delivered which result in aspiration into the bronchus of mucus and amniotic fluid, which later become an obstacle to the establishment of normal respiration. In discussing this phase of the development of asphyxia, it is interesting to note that certain investigators believe that respiratory movements are normally carried on in utero but in such manner that there is no entrance of amniotic fluid beyond the epiglottis or at most beyond the bifurcation of the trachea. At any rate, normally there is no trace of amniotic fluid in the respiratory tract of the newborn. Another point for consideration in the development of premature respiratory effort in the asphyxiated fetus is the consequent deflection of the blood stream through the right heart into the pulmonary circulation which may, by withdrawing an amount of blood from the already insufficient placental circulation serve to materially increase the degree of asphyxiation.

Pathology.—In the full-term fetus the lungs are firm, do not crepitate, and will sink in water. They are dark red and distinct septa of connective tissue stretch through the lungs in every direction. The expansion of the lungs with the onset of respiration comes about gradually, being seldom complete in the normal infant before the end of the second day, and at times even extending into weeks. Expansion first takes place in the anterior borders of the lungs then the upper lobes and last in the lower lobes. There is little uniformity in the entrance of air into the different sections of the lobes, possibly because of the blocking of some of the smaller bronchi with mucus. However, the posterior part of the lower lobes is generally last to become inflated. In premature and delicate infants owing to the feebleness of respiratory effort, areas of atelectasis are apt to persist for some time. In the infant dying from asphyxia the respiratory passages are congested, filled with mucus, amniotic fluid, and sometimes meconium. The lungs contain little air and, depending somewhat on the length of time the infant has lived, there will be more or less extensive areas of inflated lung tissue, especially in the upper lobes. Petechial and larger hemorrhages are found under the pleura and pericardium. The blood is thin and watery and there is much evidence of a general hemorrhagic status, such as multiple small hemorrhages throughout the visceral organs, extravasations of serous fluid and blood into the pleural, pericardial and peritoneal cavities. Hemorrhages in the pia mater are common. The right ventricle and large blood-vessels are usually distended. In extra-uterine asphyxia anomalies of the heart or large vessels or of the lungs will often be found. In other cases an enlarged thymus or other anomaly will cause obstruction of the trachea, or again cerebral hemorrhage interfering with the respiratory center may be the only pathological condition present.

Symptomatology.—From a clinical point of view one must keep clearly in mind the two main classifications: Congenital asphyxia and acquired asphyxia, because the underlying pathology in the two classes may be quite different. Congenital asphyxia is always preceded by fetal asphyxia. The cardinal symptoms of ante partum asphyxia are: Slowing of the fetal heart sounds, which are of especial significance if continued between pains; weakening and irregularity of the heart sounds; appearance of meconium in the amniotic fluid; convulsive movements of the fetus; and extreme rapidity of the fetal heart action due to paralysis of the vagus. This last symptom calls for immediate termination of labor if the infant's life is to be saved. When delivered, the asphyxiated infant may be cyanotic, dark-reddish or blue in color, eyes prominent, congested and apparently protruding, face swollen, body surface warm, muscular tone good and even hypertonic, heart sounds vigorous and slow, the cord pulsations strong and full. The breathing, if any, is feeble, interrupted, irregular and sometimes gasping. Cutaneous irritability is present. This gives us a picture of what we designate as first degree or asphyxia livida. In this type usually all that is necessary to establish normal respiration is the application of some external stimulus.

In asphyxia of the second degree or asphyxia pallida, the skin is pale and cold with cyanosis about the lips and the cutaneous reflexes absent. There is general muscular relaxation, the extremities hanging loosely from the trunk. The heart action is weak and may be either slow or rapid, the patient showing little signs of life; thus giving rise to the term "apparent death" so often used in this connection. Of course all the intermediate degrees of asphyxia are found between the two conditions here outlined, and the only clinical importance of the classification is to indicate the kind and amount of effort necessary to bring about resuscitation.

Acquired asphyxia is usually associated with anomalies of the heart, lungs or diaphragm, or compression of the trachea. It may be caused by increased cerebral pressure, causing anemia of the medulla oblongata thus interfering with the respiratory center and the vagus. Clinically, the infant would probably show some symptoms of these conditions responsible for the asphyxia in addition to the cyanosis, slow, weak pulse and faulty respiration. In many cases of congenital asphyxia as well as that acquired, even after resuscitation the lungs are still largely in a state of atelectasis, which is very apt to cause the asphyxia to recur, through an insufficient aeration of the blood stream.

Diagnosis.—Asphyxia fundamentally is symptomatic of underlying conditions rather than a disease in itself. The condition of congenital asphyxia might be confused with cyanosis due to cerebral compression from meningeal hemorrhage. In this case the coagulation and bleeding time might give important information. In cerebral hemorrhage the breathing might be quite strong and well established. Other signs of cerebral hemorrhage, such as bulging fontanelle, paralyses, twitchings or convulsions would point to a diagnosis. However, asphyxia is very apt to be associated with hemorrhage. It is well known that many asphyxiated babes develop multiple hemorrhages. In this connection it is interesting that the old teachers used to name asphyxia neonatorum as prominent among the causes of Little's disease, idiocy, etc. We now believe that intercranial hemorrhage is often responsible for these conditions and the problem is still unsolved as to just how much the state of asphyxia is fundamentally responsible for the hemorrhagic status.

Treatment.—The treatment indicated in this condition depends largely upon the degree of the asphyxia, the fundamental thing being to start adequate functioning of the lungs in order to reestablish the physiological oxygen-carbon dioxid balance in the blood stream. This is accomplished by removing from the mouth or respiratory tract any mucus or amniotic fluid which may be obstructing the free entrance of air into the lungs, the excitation of respiration by external stimulation, artificial respiration, and stimulation of the heart and respiratory center. Second only in importance to the establishment of respiration is the early detection of hemorrhage which is accomplished by the determination of coagulation or bleeding time. This should be done routinely during the first three or four days in all cases of asphyxia. Subcutaneous administration of whole blood should be started immediately if the coagulation time is appreciably delayed. Indeed the incidence of hemorrhage is so great in severely asphyxiated children that the routine subcutaneous administration of adult blood might be an excellent prophylactic.

In the ordinary case of asphyxia livida it is recommended to cut the cord before tying and allow from 15 to 30 c.c. of blood to escape in order to relieve the engorgement of the vascular system. In addition to this, the application of some form of stimulation to the skin, such as spanking, spraying with cold water, or rubbing with a towel is usually all that is necessary to establish deep and normal respiration. In cases in which the asphyxia is more severe, as in asphyxia pallida, all the resources and perseverance of the physician will be called upon to resuscitate the child and in many cases after resuscitation has been accomplished the asphyxia again develops usually with a fatal termination. Efforts should be continued until the heart ceases to beat which may be as long as an hour or an hour and a half, after birth, with hardly perceptible respiration. A sudden drop in an infant's temperature of from 15 to 20 degrees with complete dilatation of the pupils are given as signs that death is imminent. In cases of deep asphyxiation, before the severing of the cord, the child should be held suspended with head down and the mucus and amniotic fluid removed from the mouth and throat by means of a soft cloth wrapped about the finger. When there is evidence of fluid and mucus in the trachea and bronchi this may be aspirated by means of a soft rubber catheter. The skin should be irritated in the manner suggested for the milder degree of asphyxia. Placing the child in a hot bath of from 105 to 108°F., and suddenly transferring it for a moment to a cold bath is often successful in stimulating respiration. The hot bath is also of value because it helps to maintain the body temperature and the hot water in contact with the skin causes dilatation of the superficial capillaries, reestablishing the cutaneous circulation and relieving the congestion of the internal organs due to paralysis of the vasomotor system. It is important that the infant be kept immersed in the warm bath for as much of the time as possible while artificial respiration is being carried on.

The hypodermic injection of stimulants such as camphorated oil, and caffeine and sodium benzoate, etc., is not of as much value in the beginning in establishing respiration as are the various measures used to stimulate the respiratory center through reflex irritation of the skin. However, once the breathing is partially established and a tendency to relapse appears, the hypodermic administration of stimulants will often whip up the respiration and circulation to a marked degree. For this purpose the injection of from 5 to 10 minims of camphorated oil, and from 1/4 to 1 gr. of sodium benzoate and caffeine at the same time is recommended, the camphorated oil acting as a quick but rather fleeting stimulant and the sodium benzoate being slower in action but lasting for a considerable period. Colon flushings with a pint or more of hot water (110 to 115°F.) to which has been added 1/2 dr. of alcohol is sometimes of value. Massage of the heart serves to increase the action of that organ. This is accomplished by percussing the precordia three hundred times a minute with elastic strokes of the four fingers of the right hand.

In those cases in which the babe does not respond to slapping, cold water, etc., the persistent, intelligent and continuous application of artificial respiration is the only way to preserve life. Many different methods for carrying on artificial breathing have been developed, all of them depending for success upon the principle of regular slow alternate compression and expansion of the chest, to allow the entrance and expulsion of air. Among the methods most often used are those of Schultze, Sylvester, Prochowink, Byrd and Gaszynski. As the manipulation of these methods falls entirely into the field of the obstetrician, space will not be taken to describe them here. In carrying out any procedure of resuscitation care should be taken to avoid any unnecessary violence, employing only movements that are slow and deliberate. Especially is this true if there is any suspicion of hemorrhage, or in the presence of any broken bones. Laborde has recommended rhythmic traction of the tongue, at the rate of from 20 to 30 times per minute, to start respiration in severe cases. This method is successful in the hands of some operators and has the advantage that it may be carried on while the child is in the hot bath. Another method of more or less value is the direct mouth to mouth breathing, which consists of the physician filling his cheeks with fresh air and slowly blowing it into the mouth of the infant while the nose is held closed. Unless the infant is held just right a good deal of the air is apt to enter the stomach instead of the lungs, and also if any great amount of force is used there is likely to be permanent injury to the lung structure. There is also grave danger of respiratory infection from this method. Various methods of introducing air or oxygen directly into the lungs by means of different types of catheters have been devised, Ribemond's tube being the one most used for this purpose.

Prognosis.—In mild cases in which little effort is necessary to establish respiration, the prognosis is uniformly good. But in the severe forms many children die without responding in any way to any effort at resuscitation, and of those who do respond to extreme treatment, 20 to 30 per cent. relapse and die. The incidence of hemorrhage in these cases is high, and many who overcome the initial asphyxia die within a few days from the hemorrhagic disturbance, or live only to become paralytic or mentally defective.

BUHL'S DISEASE

(Acute Fatty Degeneration of the Newborn)

This disease of the newborn, characterized by acute fatty degeneration of the viscera, especially the heart, liver, spleen and kidneys was first described in 1861 by von Hecker and Buhl. The disease is very rare and not well defined from the other forms of acute illness in the newborn which are now attributed to general sepsis. The best contributions on the subject are by Buhl, Runge and Roach who described the condition about as follows:

Etiology.—Not much is known about the etiology, although it is now generally thought to be some form of septic poisoning. This is borne out by the fact that a similar condition is found among the newborn of certain animals. However, the disease has never been observed to occur epidemically. Many of the infants are born deeply asphyxiated and Buhl's report suggests the possibility of the infection having taken place during the last days of pregnancy. The umbilical vessels have shown no evidence of local infection so the conclusion is that the infection must gain entrance through the mouth or intestinal tract, or directly into the blood stream, without local change to the umbilical vessels. Like many other forms of septic poisoning of the newborn this condition occurs increasingly less often since the development of proper aseptic care. It is very likely that in the light of our modern knowledge of bacteriology these cases will fall into the general class of pyogenic infections.

Pathology.—The characteristic changes in this condition which appear at post-mortem examination are multiple hemorrhages with evidence of parenchymatous inflammation followed by fatty degeneration of the heart, liver, kidneys, and spleen. The hemorrhages are found in the dura and pia mater, in the pleura and pericardium and the tissues of the mediastinum, also in the thymus, the peritoneum and mucous membranes. The lungs show hemorrhagic infarcts with the bronchus filled with bloody mucus and sometimes pure blood. Hemorrhages are found in the walls of the stomach and intestines and their cavities are often filled with blood. The brain is abnormally soft and congested. The spleen is enlarged and its tissue substance so soft as to be almost fluid in consistency. The liver is swollen and tender, with marked evidence of fatty degeneration. The cortex of the kidneys is edematous and filled with blood. There are hemorrhagic infarcts throughout the parenchyma with acute inflammation and fatty degeneration of the epithelium of the tubules. The heart muscle is soft, pale and the seat of fatty change.

Symptoms.—Infants affected with Buhl's disease are usually born extremely asphyxiated. The child presents the typical picture of a case of sepsis with a marked tendency to hemorrhage from the umbilicus, nose, bowel or under the skin. Edema is often present and diarrhea is rather characteristic. There is nothing otherwise in the clinical picture to differentiate it from the ordinary case of sepsis except that in this disease there is never evidence of a local focus. The child usually dies before the end of the second week.

Diagnosis.—As has been indicated, there is nothing clinically to differentiate Buhl's disease from the ordinary case of sepsis with hemorrhage and parenchymatous changes. Microscopically the fatty degeneration will predominate in Buhl's disease.

Treatment.—The treatment should follow the same lines as in cases of septic infection.

TETANUS NEONATORUM

Tetanus in the newborn is characterized by the same symptom complex that manifests itself in older children and adults, when suffering from the same infection, the only difference being the portal of entrance, a more severe course, and a somewhat higher rate of mortality.

Etiology.—It is now well established that the organism first discovered by Nicolaier in 1884, known as bacillus tetanus, is the specific agent responsible for the disease long known by that name. The customary habitat of the germ is the soil and dust, and it is especially found in earth that has, over a long period, been intensely fertilized and cultivated. A case of tetanus in the newborn immediately presupposes the contamination of the umbilicus with the bacillus tetanus. This may occur from hands, clothing, or instruments that have not been properly sterilized. The disease seems rather restricted to certain areas where the bacillus is known to abound in the soil. The suggestion that certain races are more susceptible to the disease, as in the southern negro, is probably due to a natural lack of cleanliness in a region where the soil is contaminated with the bacillus. Infection may occur at any time from birth until the epidermal covering of the umbilical wound becomes complete, but seldom occurs after the first few days. The tetanus bacillus is seldom isolated from the umbilical wound, although pyogenic organisms are frequently discovered there when the infant is suffering from an attack of tetanus. Experimentally, it has been shown that the bacillus tetanus does not leave the original site of infection, but can usually only be recovered there during an exceedingly short period, possibly for only one or two days after inoculation. The destructive action of the disease is accomplished by the bacteria throwing off into the blood and lymph stream certain specific toxins which eventually travel by way of the motor nerves to the central nervous system, combining in some manner with the motor cells of the cord and the medulla resulting in an abnormal excitability of those centers. The incubation period, which is of varying length, depends somewhat upon the virulence of the infecting organism. The more severe cases develop as early as the first or second day after contamination.

Pathology.—At post-mortem examination, little structural tissue change is demonstrable. Hyperemia of the cord and meninges with possibly small hemorrhages is a fairly constant finding. Congestion of the lungs and distention of the right heart are usual (Holt).

Symptomatology.—The onset may occur from the second day to the third week after birth, but usually before the end of the sixth day. The first symptom is, ordinarily, a slight rigidity of the lower jaw which is precipitated by the act of suckling. In severe cases, this muscular spasm rapidly spreads over the face, soon to be followed by frequently recurring, convulsive rigidity of the whole body, which, however, is entirely relaxed between the spasms which may be brought on by the slightest movement. As the disease progresses, the convulsions become more and more frequent, with the rigidity only partially disappearing in the interval. The face assumes a fixed expression, with deep wrinkles in the forehead, eyelids tightly closed, corners of the mouth drawn down, and a rigid lower jaw. Opisthotonos may be present to a marked degree. The abdomen is of board-like rigidity, the arms flexed, and the hands tightly closed. The legs are extended, and the toes abducted. The temperature varies from a slight rise to 104 or 105°F. Owing to the rigidity of the throat, the child is unable to cry or swallow. Spasm of the diaphragm, and exhaustion are ordinarily the immediate causes of death, which may occur during the first 24 hours after onset, and usually occurs before the end of the sixth day. In the milder cases, the onset is apt to be later, and the symptoms may be limited to a slight rigidity of the jaw (Roach), however, there are as a rule recurring tonic convulsions which are of slight duration, and infrequent occurrence, with complete relaxation in the interval. In those that recover the convulsions become less and less frequent, and less severe in character until they disappear entirely, and recovery takes place.

Prognosis.—The mortality rate is estimated to be as high as from 80 to 95 per cent. It is particularly bad in those cases which develop during the first few days after birth.

Diagnosis.—The diagnosis is made from the clinical picture, and it is not likely to be confused with any other disease of the newborn, although the symptoms of cerebral hemorrhage may to some degree simulate those of tetanus.

Treatment.—Tetanus, always a rare infection in the newborn, is practically unheard of except in certain localities, among people who have failed to appreciate the value of rigid cleanliness and asepsis. Once an infection occurs, treatment is of little avail except as it may possibly ameliorate, to some extent, the keen distress suffered by the infant. Tetanus antitoxin has now come into regular use as a prophylactic measure, and should be used freely at the earliest, slightest suspicion of infection. When used within the first few hours after contamination with tetanus, we have learned to look upon this treatment as specific. However, once the disease has developed, antitoxin seems to have little curative value. This is explained by the fact that the serum, when injected into the system, instead of destroying or inhibiting the growth of the tetanus bacillus, accomplishes its purpose by combining with the toxins as they appear in the blood stream, thus neutralizing the poison before it has an opportunity to invade the central nervous system. However, when the first symptoms of tetanus appear in the infant, the toxins have already attacked the motor cells, and the antitoxin is apparently unable to disturb their activity. For this reason, the only thing to be hoped for, by the injection of antitoxin after the disease has developed, is that there may still be quantities of toxin appearing in the blood stream which may be neutralized by the serum. It is now known that much larger doses of the antitoxin may be given safely than was formerly supposed. Doses of from

1000 to 2000 units should be given repeatedly during the course of the disease. To control the spasms, chloral hydrate is the most reliable drug, a dose of 5 gr. in aqueous solution by rectum may be given repeatedly up to 45 to 60 gr. per day. This may be given in combination with calcium bromid in doses of from 15 to 30 gr. Other drugs that have been recommended are hypodermic injections of atropin sulphate, in doses of from $\frac{1}{1000}$ to $\frac{1}{3000}$ gr. per day. One-tenth grain of the extract of calabar bean may be given hypodermatically from 3 to 10 times daily. The rigidity of the jaw interferes with the nursing, but every effort should be made to maintain the nutrition of the infant. Breast milk must be expressed and fed to the baby by a spoon or medicine dropper, and when the spasm around the mouth will not allow this, a soft English No. 6 rubber catheter should be passed through the nose, and the milk introduced into the stomach in this manner. As noise and movement are likely to precipitate spasms, every attempt should be made to keep the infant as quiet as possible. Rigid isolation procedures must be carried out.

DISEASES OF THE UMBILICUS

Disturbances of the umbilical region in the newly born which interfere with the normal physiologic obliteration of the umbilical cord and blood-vessels of primarily prenatal function, fall into two general divisions: First, those due to anomalies, malformations and tumors, second, those due to infections.

Under ordinary conditions, with the advent of respiration, the flow of blood into the pulmonary circulation greatly lowers the blood pressure in the circulation of the cord, which is followed by the collapse of the umbilical arteries and veins, and a discontinuance of placental circulation. The large amount of muscular and elastic tissue in the walls of the umbilical vessels so effectually constricts the lumen, in connection with the drop in blood pressure, that in most cases even if the cord were not ligated little if any hemorrhage would occur. The vessels of the cord have no capillaries and thus with the discontinuance of circulation, atrophy and mummification of the cord and vessels take place. A sharp line of demarcation, attended with slight inflammatory reaction, occurs at the junction of the amnion and skin of the navel, separation of the umbilical stump usually being complete by the fifth or sixth day. This is followed by a folding in of the skin over the umbilical wound which is retracted owing to the shortening and connective-tissue proliferation of the intra-abdominal umbilical blood-vessels. At the point of separation of skin and amnion, proliferation of the epidermis occurs and continues until the umbilical wound is covered with skin which is usually accomplished by the end of the third week. The intra-abdominal portions of the cord vessels rapidly undergo change and are gradually converted into connective tissue, the arteries becoming the lateral umbilical ligaments and the vein becoming the round ligament of the liver.

Class I conditions nearly always result from accidents or perversions in embryonic development. Prominent among these are amnion navel, cutis navel, hernia of the umbilicus or of the umbilical cord, persistence of the omphalomesenteric duct, urachus fistula, and congenital nipping off of umbilical hernial protrusions. Tumors occurring in this region, most of which are very rare, are angiomata, myxomata, fibromata, papillomata, lipomata, and occasionally dermoid cysts.

Amnion Navel.—This is a rare condition characterized by the extension of the amniotic epithelium upon the abdomen from the cord, replacing only the skin normally present around the umbilicus; the subjacent fascia, abdominal muscles and peritoneum being normal in the majority of cases. As a rule the amniotic navel is of small area and does not necessarily interfere with the health of the child, other than predisposing to the development of umbilical hernia. The skin defect usually promptly heals by granulation and scar formation. In extreme cases the area is quite extensive, the normal structures composing the abdominal wall may be entirely absent and the abdominal contents covered merely by a thin transparent layer of the amnion through which the viscera are plainly visible. Due to the weakness of this covering marked hernial protrusions of the abdominal viscera occur. In these extreme cases immediate surgical procedure offers the only chance of saving the life of the child.

Cutis Navel (**Skin Umbilicus**).—Under normal conditions the junction of the epithelium of the skin of the child, and that of the amnion is marked by a very definite line of transition encircling the cord at a ring-shaped swelling approximately 0.5 cm. to 1 cm. out on the cord. In the case of cutis navel the epithelium of the skin extends a greater distance than normally, forming a skin-covered cylindrical protuberance, on the end of which is found the umbilical wound. Ordinarily after the wound heals the projection remains. Occasionally retraction occurs, but when it does not, healing is often delayed owing to excoriation from dressings, binders, etc. The tumor disappears spontaneously as the child grows older, the excess skin being taken up in the general abdominal development. When retraction does take place it is likely to produce a deep navel fold in which granulomata are prone to develop.

Umbilical Hernia.—By a process as yet not completely explained, normal indentation of the umbilicus begins by a rolling in of the skin, first along the lower margin of the navel. This change continues slowly until the umbilication, finally, is completed shortly after birth. Not infrequently, however, the process is considerably delayed, therefore a small umbilical hernia is a very common and not especially abnormal finding in infants. The protrusion occurs usually at the so-called "umbilical weak spot," ordinarily situated to the right of the umbilical vein, and above the umbilical arteries. The hernia is small in the majority of cases, measuring 1 to 2 cm. in diameter, and tends to decrease in size as the infant grows older, even if untreated. Spontaneous cure may be favored and hastened by reducing the hernia manually, and maintaining its reduction with a broad strip of adhesive applied over the umbilicus in such manner as to hold the skin and subcutaneous tissues on each side of the navel together in a deep fold, the groove of which should run parallel to the linea alba. The rarity of the condition in adults, which is so prevalent in children, is evidence of the tendency for the condition to disappear spontaneously. Only very exceptional cases require surgical attention.

Hernia of the Umbilical Cord.—During early fetal life a considerable portion of the small and large intestine normally lies in the umbilical cord or body stalk occupying the exocelomic cavity. Under normal conditions the intestine gradually recedes into the abdomen, and the cavity in the cord is obliterated. Occasionally, however, this process fails to be completed, and in such instance at birth a considerable portion of the intestine and possibly other abdominal viscera may be found occupying a cavity in the fetal end of the cord. The mass varies in size from that of a hazel nut to that of a child's head and may be either narrow or broad at the base. The covering of this hernia is a thin semi-transparent membrane composed largely of amnion and peritoneum in which are occasional disseminated masses of Wharton's jelly. In cases in which the mass is large the hernial sac is sometimes ruptured during birth. In this type of hernia there is little or no tendency toward spontaneous repair, and because of the great danger of rupture or infection surgical treatment should be instituted at once. In case of small hernia cure may be effected by merely tying off at the base, but before this is undertaken great care must be taken to make certain that a loop of intestine is not strangulated by the ligature. When the hernia is more extensive a radical surgical operation is indicated.

Congenital Nipping Off of the Umbilical Hernial Protrusion.—During fetal life, while the intestines are withdrawing from the exocelomic cavity within the umbilical cord, in rare instances the intestines may become adherent to the cavity and are thus prevented from withdrawing into the abdomen. Under such circumstances the umbilical ring may close and as a result loops of the intestine may be nipped off forming a tumor mass in the region of the umbilicus. On examination of the intestines in this condition, the lumen is found to gradually narrow at the umbilicus, and to end blindly at the point where the hernial protrusion passes through the abdominal wall. The intestine thus becomes continuous only by means of a fibrous cord which connects its upper and lower, apparently normal portions. In these cases the infant manifests the classical signs of intestinal obstruction with a failure to pass meconium. Should a diagnosis be made and the condition of the infant give any hope of its standing the operation, an attempt at surgical repair should be made.

Persistence of the Omphalomesenteric Duct.—Remnants of the omphalomesenteric duct, producing a secretion at the umbilicus, constitutes one of the most interesting groups of pathologic conditions found in the umbilical region. The most common remnant of this duct is a small polyp located in the umbilical pit. It is bright red and secretes a substance of mucus consistency. Microscopic examination shows the surface of the polyp to be covered with a mucosa very similar to that of the small intestine, and in rare instances resembling that of the stomach, and in this case it secretes a serous fluid very similar to gastric juice. In other cases the umbilical discharge may come from a patent omphalomesenteric duct or Meckel's diverticulum, instead of being secreted from the mucosa covering a small polyp at the umbilicus. In this case fecal matter may be mixed with the discharge. The amount of discharge varies, with a tendency to increase at the time of eating. These conditions are easily amenable to surgical treatment.

Urachus Fistula.—In exceptional instances the remnants of the stalk of the allantois extending from the umbilicus to the summit of the bladder and known as the urachus contains only a partially obliterated lumen. After birth when, through some obstruction of the urethra, the bladder becomes greatly distended, urine occasionally finds its way through the urachus and discharges at the umbilicus. At the mouth of the fistula a small red tumor may be found similar to that noted in the case of persistence of the omphalomesenteric duct. This tumor, according to Pfaundler and Schlossmann, owes its origin to a prolapse of the mucosa lining the urachus. Cauterization of the fistulous opening or suture of the walls of the fistula after freshening their surface is the treatment usually recommended.

Angiomata of the Umbilicus.—This type of tumor is congenital in character, and in some cases is considered as originating from a remnant of the omphalomesenteric duct. On microscopic examination the tumor, like angiomata elsewhere, is found to be composed of dilated blood-vessels and connective tissue. These tumors are benign and the treatment is surgical if any be needed.

Myxomata of the Umbilicus.—This type of umbilical tumor is exceedingly rare, only a very few cases being reported in the literature. When the gross specimen is examined these tumors are seen to be white, soft, pale, edematous, and gelatinous. The size varies from that of a bean to that of an egg. Microscopic examination shows the tumor to be composed of numerous blood-vessels embedded in a connective tissue, consisting mainly of large spindle-shaped cells, between which is a ground substance similar to that found in embryonic tissue. It has been suggested that these tumors may originate from remnants of Wharton's jelly. As a rule the tumors are covered with normal skin, although in some instances the surface is ulcerated. The tumors are benign, hence the prognosis is good.

Fibromata of the Umbilicus.—These tumors are exceedingly rare in infants. As a rule they are rounded, pedunculated tumors attached to the umbilical depression. On section the tissue is seen to be benign in character and composed of connective tissue covered with normal or slightly atrophic, epithelium. Areas of cystic degeneration containing serous fluid may be found within the tumor.

Papillomata of the Umbilicus.—Tumors of this character have been reported in young infants. On section the tumor consists of a core of connective tissue covered with stratified squamous epithelium, and resembling skin papillomata in any other part of the body.

Lipomata of the Umbilical Region.—Since there is practically no fat present at the umbilicus, lipomata in this region are very unusual. When the umbilical ring is rather large, however, some of the subperitoneal fat may slip through the umbilical ring and produce an adipose hernia or lipoma of the umbilicus.

Dermoid Cysts of the Umbilicus.—True dermoid cysts of the umbilicus are very rare, only some half-dozen authentic cases having been reported in the literature, all in young children. The cyst is usually pedunculated, attached at or near the umbilicus and contains a fatty sebaceous material. The cysts are lined internally with stratified squamous epithelium devoid of hairs and glands. In none of the cases have hairs been detected in the cyst contents. The skin covering the tumor is usually intact. The tumor is benign.

Umbilical Infections.—Of diseases of the umbilicus, Class II comprises those arising from contamination and infection. They are further divided, for the purpose of classification, into, first, those which are ordinarily local in character and, second, those which are often the basis of general systemic infections.

In the first class we have such mild conditions as delayed healing, blennorrhea, ulceration and fungus, and circumscribed omphalitis, while in the second division are grouped gangrene of the umbilicus, phlebitis and arteritis of the umbilical blood-vessels, tetanus, and erysipelas, and occasionally gonorrheal or diphtherial invasion.

The eternal watchword in everything pertaining to the care of the umbilical cord and umbilicus of the newborn is "asepsis." The style of dressing or the materials employed is of little consequence if every possibility of infection be avoided by the most rigid adherence to the principle of asepsis. As a rule, the umbilical cord should be tightly ligated as soon as it has ceased to pulsate, advisedly by two sterile ligatures, the first about 1 cm. from the abdominal wall, the second some 2 cm. from the first. The cord is incised with sterile scissors, between the two ligatures. Dry dressings should invariably be used, of sterilized gauze, or absorbent cotton, with powdered boric acid, as a wet dressing delays the mummification and sloughing off of the cord which should normally take place within the first week. At the point of separation is a granulating wound, which should be treated as any fresh wound with every surgical precaution. If suppuration occurs the umbilicus should be cleansed daily with bichlorid solution or alcohol, and fresh sterile dressings applied. Any unnecessary manipulation that might induce hemorrhaging should be avoided. Infection may take place through the umbilicus either before or just after the separation of the stump, and until the epithelial coating is formed, the child should be very carefully bathed, that the umbilical dressing may not become contaminated. Pyogenic infection in the newborn is a serious and a preventable disease. Only eternal vigilance on the part of the physician and nurse in attendance can protect the infant against this bacterial invasion, which in any but its lightest forms almost always proves fatal.

Delayed Healing.—After ligation of the cord, the stump if kept dry usually undergoes mummification but when the area is kept moist a moist gangrene of the stump often results, with active inflammatory processes soon producing a diffuse redness which may spread gradually over the entire skin of the umbilicus. Leukocytes collect around the dead stump and gradually soften and loosen the necrotic tissue, so it falls off usually on the fifth day postpartum. This separation of the stump occurs somewhat later in premature and weak infants. The amnion separates first, then usually the arteries, and lastly the vein. After the cord drops off normally the local inflammatory signs gradually subside, and healing is usually complete by the end of the third week. Delayed healing of the wound may result from a large variety of causes including mild and severe umbilical infection, presence of remnants of the omphalomesenteric duct, fistula of the urachus, fungus and other conditions.

Normally, there is a slight inflammation around the region of the umbilical stump, so that it is difficult to determine accurately the transition from the healthy to the slightly infected umbilicus. The presence of the infection delays healing of the wound for a variable length of time, and increases the amount of discharge accumulating about the umbilicus. In the less severe infections the area of the umbilical wound may increase somewhat, and may be covered by a layer of whitish necrotic tissue and exudate, but usually responds promptly to treatment and does not affect the normal progress of the infant.

Blennorrhea.—After the umbilical stump separates, a seropurulent discharge sometimes arises which may persist for some time. The necrotic tissue remains of the umbilical stump affords a very excellent medium for bacterial growth especially when moist. It is in such instances that infections produce a rather abundant discharge from the umbilical wound. The umbilicus itself may become slightly reddened, and the base of the umbilical indentation filled with red secreting granulations. This condition is spoken of as blennorrhea or catarrhal omphalitis. The infection often finds its way into the thrombi of the umbilical arteries thus forming a deep fistula which constantly discharges pus into the umbilical wound. This form of invasion rarely becomes systemic and usually responds readily to treatment although deep opening of the fistula is sometimes necessary. The diagnosis of blennorrhea is established when the presence of a patent omphalomesenteric duct, diseases of the umbilical vessels, and umbilical gangrene are excluded.

Omphalitis.—This condition, often described as acute umbilical cellulitis, occurs in all degrees of severity from a slight edema, redness, and swelling of the tissue immediately surrounding the umbilicus, due to extension of a mild infection of the umbilical wound, to a generalized cellulitis and lymphangitis with general toxemia occasionally leading to peritonitis and general sepsis. In mild cases little other than local symptoms of redness and swelling are seen with sometimes a purulent discharge from the navel while in the more severe forms there is a general invasion of the abdominal wall which is hard and indurated from extensive infiltration in the region of the umbilicus, which may protrude markedly. As the process continues the skin becomes tense, edematous, and very tender, the inflammation extending out over the abdomen and down over the pubes, the lymphatics if involved showing as fine red streaks around the umbilical region. An accumulation of pus occurs under the umbilical swelling. Breathing is diaphragmatic in character, there is a general rise in temperature, and the infant gives every evidence of acute distress, the legs being usually flexed over the abdomen. Free incision should be made at the umbilicus to insure ample drainage and with warm, moist antiseptic compresses, will often effect a cure even in cases of seemingly extensive involvement.

Umbilical Ulcer.—Delayed healing of the umbilical wound is often caused by the development of an area of ulceration which may be very small or extend to be as large as 2 cm. in diameter. They frequently arise as the result of infection with one of the pyogenic organisms, and are pea-size or smaller in character. They are usually confined to the umbilicus with the inflammatory reaction extending into the skin of the navel folds. The center of the ulcer is often covered with a thick white exudate and surrounded by a sharply defined, indurated edge. It has been noted that the stump of the umbilical artery often appears in the center of the necrotic area. The ordinary dry antiseptic dressing and cauterization with silver nitrate is usually sufficient to effect prompt healing of the ulcer.

Umbilical Fungus.—This fungus takes the form of a granulomatous tumor which not infrequently arises from the umbilical wound and delays the ordinary healing process. The tumor varies in size from that of a millet seed to a hazel nut, but is seldom larger than a pea. The surface has a red, raw, lobulated appearance, secretes a serous fluid, and the umbilical wound refuses to heal. The presence of the tumor is often not discovered until the skin of the umbilical fold is held open when the granuloma may be seen attached to the umbilical wound. These tumors are very vascular in character but have no nerve endings. All but the largest ones can be successfully removed by cauterizing with silver nitrate. If snipping off is attempted it is wise to at first ligate the base because of the marked tendency toward bleeding.

Umbilical Gangrene.—Gangrene of the umbilicus is a condition seen much less frequently now than formerly. It is more prone to occur in premature and poorly nourished infants, and especially in association with omphalitis, umbilical ulceration and infection, and also in association with general sepsis, especially following cholera infantum. With the development of gangrene the area about the umbilicus becomes discolored, indurated, and large blebs with cloudy contents may develop. The discolored gangrenous area enlarges rapidly, tending to spread down and producing large sloughs of gangrenous, necrotic tissue. As a rule, the body temperature is increased little if at all. The odor from the gangrenous tissue is very foul. As a rule, gangrene of the umbilicus occurs only in newborn infants. However, in the type associated with cholera infantum or general sepsis, this form of gangrene may appear in children several months of age. If the gangrenous area is small, recovery occasionally takes place, but in the more severe cases peritonitis usually develops to be followed by general sepsis and death.

Inflammation of the Umbilical Vessels.—Arteritis, periarteritis, phlebitis and periphlebitis are practically always secondary to an infection of the umbilical wound which has invaded the region around the umbilicus and spread out along the blood-vessels. There has been much dispute as to whether the infection of the vein or artery is the more frequent and also as to which infection is the more serious. At present, however, it is generally conceded that umbilical arteritis and periarteritis are the more common and likewise are more benign. Many cases of arteritis occur without symptomatic evidence. The inflammation usually starts in the adventitia in both the vein and the arteries so a primary periarteritis and periphlebitis usually exist which extend into the lumen and infect the thrombi. In the arteries is a large amount of perivascular connective tissue, which is not found in the vein, and the fact that this connective tissue is considered an excellent media for bacterial invasion is offered as the explanation for the more common occurrence of arteritis. There is no characteristic group of symptoms arising primarily from the infected umbilical vessels. In many cases the umbilical wound may be entirely healed, and especially in a case of arteritis, when the infection only involves a part of the vessel, the disease may run its course without having given rise to any appreciable disturbance. Occasionally, when the vessels are infected at the end nearest the umbilical wound the navel may be covered with crusts or there may be an oozing of pus. In the severe cases we may have a slight rise in temperature, distention of the abdomen followed by peritonitis, and occasionally a general sepsis. Postmortem, examination usually shows the umbilical arteries and surrounding tissues to be the seat of an acute inflammatory process, which extends into the peritoneum. Other common findings are, besides the peritonitis, purulent or serous pleurisy, pneumonia, subcutaneous abscesses, phlegmons, periostitis, osteomyelitis, infarcts in the spleen and kidneys, nephritis, purulent meningitis, cerebral hemorrhages, and encephalitis (Pfaundler and Schlossmann).

The prognosis in arteritis is good except in those cases in which peritonitis or general sepsis develops. There is little to offer in the way of treatment, except prophylactic measures. Phlebitis or periphlebitis are thought to occur less often because of the greater resistance of the wall of the vein. This condition is usually accompanied by arteritis and seldom gives distinctive local symptoms, pus rarely discharging from the vein into the umbilical wound. The clinical signs are only those which arise from an extension of the inflammation from the vein into the liver and the peritoneum and the development of general sepsis which almost always follows. It is in this type, particularly, that we find a bloody oozing from the umbilicus and a profound icterus. In any but the very mildest forms of infection of the umbilical vein the outcome is invariably fatal.

THE NERVOUS SYSTEM

Disturbances of the nervous system of the newly born infant are generally the result of injury incurred during the birth process, but may occasionally be of congenital origin, or in certain cases the result of toxicity. The nervous system of the newborn is essentially very unstable, slight stimulation producing extraordinary manifestations of hyperirritability. While some infants are relaxed and their reactions not easily excited, larger numbers will be observed with a general muscular hypertonicity, and a nervous system of extremely high tension. In these latter cases a relatively small disturbance may result in a general nervous explosion, which may explain why so many diseases or injuries of the newborn are frequently accompanied by convulsions.

Convulsions

Convulsions, and increased temperature, are the most common symptoms of any serious disturbance in the newborn. Ordinarily the infant in convulsions is suffering from some injury or disease, the source and cause of which can usually be determined by proper investigation. As more and more progress is being made in the study of the newborn we are learning that convulsions are seldom idiopathic but are symptomatic of some demonstrable pathological condition.

Cerebral hemorrhage, either due to injury at birth or the result of hemorrhagic disease, is responsible for more cases of convulsions in the newborn than the sum of all other causes. Any infection or other condition which produces high temperature is not infrequently accompanied by convulsions. Eclampsia in the mother not uncommonly results in convulsive seizures in the child. Other causes to be considered are meningitis, spasmophilia, tetanus neonatorum, and epilepsy.

Injuries to the head causing cerebral hemorrhage, and hemorrhagic disease of the newborn have been discussed in another chapter and it is only necessary to mention here that convulsions occurring in the first four days of life are usually the result of cerebral hemorrhage in one form or another. Febrile disturbances in the newborn are often accompanied by convulsions which discontinue when the fever subsides. The so-called inanition fever which usually occurs only in infants who are markedly dehydrated and suffering from lack of food, may produce an extremely high temperature and be accompanied by severe convulsive seizures. Septic infections of various types are often the cause of spasms occurring subsequent to the onset of the disease. This has been more fully discussed under the head of "Sepsis in the Newborn." With the marked decrease in the number of cases of tetanus neonatorum convulsions from this disease are rare. This condition has been discussed under tetanus neonatorum.

It is not uncommon for the infant of a mother suffering from acute nephritis to develop convulsions immediately following or within the first two days after birth. The severity of the eclamptic seizures in the mother are no criterion of the amount of toxemia in the infant. A great many children born of mothers having severe eclamptic convulsions just before or just after the delivery, show no signs of toxemia, while often in cases where the mother is only mildly toxic the infant may be extremely so. The children of these eclamptic mothers are often of very low vitality and occasionally are still born. The convulsions may occur as early as two or three minutes after birth and the onset is seldom delayed beyond the first 48 hours. In arriving at the cause of the disturbance in these cases the history of the condition of the mother usually leads directly to the diagnosis. There is seldom any marked renal irritability in these cases, and the prognosis is favorable for rapid subsidence of symptoms. The convulsive seizures are due to toxins in the infant's circulation which have been absorbed from the mother's blood.

Meningitis is extremely rare as a primary infection in the newborn and convulsions due to this cause would be present only after the history of a febrile disturbance and very likely some other clinical evidence of the infection.

Spasmophilic diathesis if ever present during the first three months is usually latent, as the classical signs of tetany are rarely demonstrable during the newborn period. The electrical reactions in the newborn are customarily, under normal conditions, the same as are found in later infancy, and the classical electrical reactions of spasmophilia are never present. Neither do we find the laryngospasm so often seen at a later time. Therefore, convulsions of the newborn appear to be seldom, if ever, the result of spasmophilic diathesis.

Epileptic convulsions in these young infants are usually simply the manifestation of cortical irritation due to a comparatively small hemorrhage or injury to the brain and are seldom idiopathic in origin.

Routine treatment of convulsions in infancy has been covered in the chapter on intracranial hemorrhage.

Congenital Organic Disease of the Nervous System

A fairly large number of abnormalities and deformities of the central nervous system of congenital origin are well known and have been many times reported and described in the literature. Among these we find acephalia (amyelia) anencephalia (hemicephalia), porencephalia, microcephaly, congenital hydrocephalus, encephalocele, and meningocele. Some of these, such as amyelia, are never compatible with extra-uterine life, while infants afflicted with other abnormalities, such as anencephalia, may live from a few minutes to two weeks. From a clinical standpoint these conditions are of interest only because of the study they present of the abnormal development of other parts of the body, a discussion of which comes more within the field of the neurologist and pathologist than that of the pediatrist.

Acephalia (amyelia) is a condition in which there is a total absence of the brain and cord. Such cases rarely survive the early fetal period.

Anencephaly is the term used to describe the congenital absence of both cerebral hemispheres. It is probably the most common of the brain mal-formations and has been known to occur more than once in the same family. The calvarium is usually absent, the cranium is enclosed by a dark-red, irregularly shaped mass of vascular tissue. The medulla and cord are as a rule well developed and the general muscular and skeletal tissues are normal. Abnormalities of the suprarenal glands have been reported, and the writer has observed a recent case, in which the adrenals were seemingly replaced by a fatty degeneration, the thymus was tremendously enlarged, and the thyroid about twice the normal size. The etiology of this condition is obscure but is suggested to be the result of cleft formation in early fetal life, or of failure of the medullary tube to close. As stated above the condition is not compatible with extra-uterine life of more than a few hours or days.

An **encephalocele** is a hernia of a portion of the brain together with the meninges through some defect in the bony structure of the skull. When the hernia contains only the meninges, it is known as a meningocele and when it includes ventricular fluid it is described as a hydroencephalocele. The three usual forms of encephalocele are the basal, the frontal, and the occipital, the last of which is by far the most common. In the first, the hernia appears in the nasal or pharyngeal cavity, in the second it protrudes between the frontal bone and the nasal process, while in the last variety the hernia is situated just below or above the external occipital protuberance. The etiology is purely a matter of supposition. The tumors vary in size from a walnut to that of a child's head. They may be covered by normal skin or the covering may be very thin and shiny and of a bluish color. The base is sometimes broad, sometimes pedicular. The tumor may be of such size as to interfere with the birth of the head. The prognosis depends upon the size, the location, the type, and the structures involved. Surgical treatment is usually indicated in these cases and should be undertaken early if the tumor is of operable type.

Spina Bifida

Spina bifida is the term applied to a hernia of part of the contents of the spinal canal occurring through a congenital cleft in the vertebral wall. This is the most frequent form of deformity encountered in the central nervous system of children and is said to occur as often as once in every 1000 births. In one series it was found once in every 630 births. According to Gale, 23 per cent. are sacral, 24 per cent. lumbar, 29 per cent. lumbar sacral, 4.5 per cent. dorsal, and 9.5 per cent. cervical. The opening in the spinal canal which varies in breadth and length is supposed to be the result of failure of the medullary tube to close at some point during early embryonic life.

There are four general types of spina bifida: First, myelocele; second, myelocystocele; third, meningocele; and fourth, occulta.

Myelocele.—This is the most common and severe form of the spina bifida. The accumulation of fluid on the ventral side of the canal at the point of opening forces out the entire contents of the canal through the aperture, forming a cystic tumor with a dark-red, moist vascular covering. These tumors vary in size from a marble to a large orange, have a broad base, vary in shape, but are usually oval with an irregular surface. They are commonly fluctuating, often tense, but sometimes soft and flabby. Occasionally, the firm remnants of the cord structure can be felt upon palpation. If the surface of the tumor has not become eroded and infected, it is sometimes possible to make out the three zones which correspond to the structures forming the hernia, the zona medulla vasculosa containing the remains of the nerve elements, just under which will be found the zona epithelo serosa, corresponding to the pia, and beneath this lies the zona dermatica formed of thick skin usually very vascular and sometimes hairy.

Myelocystocele.—In this form of spina bifida, after the spinal marrow has closed, the central canal is dilated at the place of opening in the spinal canal from the internal pressure of spinal fluid, finally resulting in a protrusion through the opening of a hernial sac containing spinal fluid, the dorsal portion of the spinal marrow, and the pia, with the skin covering usually intact. In size and shape and location, this type is very like the myelocele.

Meningocele.—This type of spina bifida is a sacculated protrusion of the leptomeninges most often occurring in the cervical region. The skin covering is usually intact and when it is found in pure form, which is rare, the contents of the hernia consist of spinal fluid and the pia. It is usually pedunculated and may grow to reach a size of 5 or 6 in. in diameter. It gives no symptoms, rarely ruptures or becomes infected.

Spina Bifida Occulta.—Spina bifida occulta, as the name would indicate is that type of spinal hernia which, because of the absence of spinal fluid pressure, does not protrude and only is discovered because of the symptoms produced. It occurs in the sacral region and although, as stated above, there is no protrusion of the contents of the spinal canal, there may be a serious abnormality in the nerve structures.

Symptomatology.—The symptoms produced by spina bifida vary somewhat with the type and location of the hernia. In the myelocele and the myelocystocele the nerve structures are usually so involved as to produce diminished or lost sensation in the lower extremities, with partial or complete paralysis of the parts receiving their nerve supply from the affected portion of the cord. There is usually incontinence of urine and feces and possibly prolapse of the anus. In spina bifida occulta the symptoms are much the same in character, although of much less degree and may possibly not manifest themselves until early childhood. Spina bifida is often accompanied by other abnormalities such as hydrocephalus, and club foot.

Prognosis.—The prognosis, while uniformly bad in spina bifida, depends somewhat upon the size, type and location of the malformation. In the meningocele of the pure type, the outlook is fairly good as they seldom rupture or become infected and lend themselves readily to operative treatment. There is, however, grave danger of hydrocephalus even in this type. In myelocele and myelocystocele the prognosis depends somewhat upon the amount of paralysis. If this is great and especially if hydrocephalus is present there is little hope for improvement. Death usually results from rupture of the hernia with too rapid draining off of the spinal fluid or from meningitis due to the almost inevitable infection of the hernial mass.

Treatment.—When the spina bifida is not completely covered by skin the mass must be cared for with the most rigid aseptic technique. Dry antiseptic powder and sterile gauze dressings are recommended, as there is the greatest danger of infection followed by sloughing of the mass. The only satisfactory treatment is surgical and this only meets with success in the most favorable cases, as it brings neither relief nor cure when there is a marked degree of paralysis or hydrocephalus. As has been pointed out by one author, in those cases which survive to the age of five years, a successful operation is ordinarily possible, because it is only the mild cases with little paralysis that live to this age. For the actual operative indications and technique reference should be made to surgical texts. It is well to remember that while it is generally conceded that early operation is desirable, even in the most favorable cases a fatal hydrocephalus is rather to be expected than otherwise.

Encephalitis

Encephalitis of the fetus or newborn is a condition of which there is little known clinically. Fetal encephalitis is often the cause of various forms of aplasia and dysplasia of the brain substance, changes being sometimes gross as in the case of anencephalia or porencephalia or they may be microscopical in character as those which affect the nuclei of the cranial nerves. Congenital hydrocephalus is commonly supposed to be the result of fetal encephalitis and meningitis. The etiology is very obscure except when syphilis has been demonstrated. Suppurative encephalitis may occur accompanying meningitis but is seldom recognized until post mortem examination.

Meningitis

The cause of meningitis during fetal life is as obscure as is that of encephalitis. However, septic meningitis of the newborn is gradually becoming a little better understood and more often recognized. Koplic believes the disease escapes early attention because of its insidious onset and because the usual leg and neck signs are absent until very late in the disease. In a series of 12 cases he found the streptococcus in four, the pneumococcus in three and the meningococcus in one. Meningeal invasion is usually secondary and the presence of convulsions may be the only characteristic symptom. While some cases reported show retraction of the head, bulging fontanelle, fever and convulsions, others have been reported to run the entire course without temperature or other characteristic signs of meningitis except convulsions. Spinal puncture is the most likely method of arriving at an early diagnosis. As far as we know, no recoveries have occurred.

Congenital Hydrocephalus

Congenital hydrocephalus is practically always of the internal type although the external type has been known to occur. The etiology is very obscure but it is generally believed to be the result of an inflammatory process such as a serous meningitis or a meningoencephalitis of unknown origin. Syphilis is not now considered as important an etiologic factor as previously. The amount of interference with the brain development depends upon how early in fetal life the inflammatory process sets in. According to von Reusse the external circumference of the skull is not necessarily enlarged and may be abnormally small, presenting the characteristic shape of microcephaly. Ordinarily, if the increase in cerebral fluid has commenced before birth there will be a characteristic enlargement of the head with widely opened sutures and fontanelles. In many cases the characteristic shape of the head as seen in later infancy will be present at birth and the size may be so great as to interfere with delivery which can only be effected after cranial puncture, following which the child may be born and still be viable. The diagnosis of internal hydrocephalus in the newborn in the absence of an enlarged head is sometimes difficult and may easily be confused with intracranial hemorrhage or meningitis. Spinal puncture and transillumination of the skull are suggested as methods to be used in diagnosis. The prognosis during the newborn period is very doubtful, and the only treatment to be recommended is repeated spinal punctures which are thought to have been of benefit in some instances.

Congenital Diseases of the Spinal Cord

Congenital diseases of the spinal cord are very rare and of very obscure origin, amyotonia of Oppenheim being the only well recognized form of muscular paralysis present at birth which is thought to be due to a congenital change in the substance of the cord. These changes are, however, comparatively slight, there being some doubt in the minds of pathologists as to whether they are not secondary to the arrested development of the peripheral nerve-end plates. In this condition, which has been seen by the writer in one instance in two children of the same family, and in another in twins, the paralysis is usually unrecognized during the newborn period, as the lack of muscular tone and movement of the extremities is not likely to attract attention as anything abnormal. The picture presented is that of a child of normal development with extreme muscular weakness which is usually more evident in the legs, but to a lesser extent also involves the arms and to some degree the body and neck. The reflexes are absent or diminished in direct proportion to the paralysis. The prognosis depends somewhat upon the degree of involvement, and in the very mild cases it is possible that some improvement will occur, life being prolonged indefinitely. In the more severe cases, however, little improvement is to be expected and death from some intercurrent disease, usually pneumonia, may be expected during the first three or four years.

INJURIES TO THE EYE

Owing to the natural protection to the eyeball offered by its bony surroundings the occurrence of direct injury during birth is very unusual and this is particularly true in spontaneous delivery. Severe injury, however, has resulted from the faulty application of forceps on the unengaged head. Practically no traumatism to the eye structures occurs except in head or face presentations.

Swelling and edema of the eyelids, usually bilateral, are often present following a face presentation forming a part of the so-called caput succedaneum. Ordinarily this swelling subsides rapidly without much damage and treatment is unnecessary.

After difficult forceps delivery, especially when one blade of the forceps crosses the eye, superficial abrasions and traumatism of the lids are frequently seen. The injured lid becomes much swollen and discolored, and occasionally may be so cut or torn that suturing becomes necessary to prevent ectropion.

Hemorrhages into the optical conjunctiva are very common and are the result of extravasation of blood from the rupture of minute conjunctival blood-vessels during the progress of labor. Such hemorrhages are rather slow to absorb but usually disappear by the end of the third week. They leave no permanent defect and require no treatment.

As has been stated before, injury to the eyeball is very unusual during natural delivery, although the dislocation of an eyeball, the rupture of ocular muscles, and hemorrhage within the structure of the eyes have been reported. As previously stated severe injuries to the eyeball, the ocular muscles, or the optic nerve may be brought about in the application of forceps, but in such cases it is almost necessary to fracture the bony structures of the orbit before the soft parts will come to injury.

CONJUNCTIVITIS OF THE NEWBORN

Synonyms.—Conjunctivitis neonatorum, ophthalmia neonatorum, blennorrhea neonatorum.

The term ophthalmia neonatorum is academically a generic name for all affections of the conjunctiva of infants, characterized by certain symptoms in common, but does not designate an etiologic entity. It has come to be, however, more specifically the name for the purulent inflammation of the conjunctiva caused by the Neisserian infection. Hence, when the term ophthalmia neonatorum is used in designating any infantile conjunctivitis a gonorrheal or allied inflammation is usually understood.

Histology.—At birth the conjunctiva is sterile, with the very rare exceptions, which follow intra-uterine or hematogenous infection. It is an almost colorless membrane with a moist luster and so transparent that through it the Meibomian glands are seen as yellowish-white thread like lines in the tarsi. The ocular conjunctiva cover the white or bluish-white sclera. The surface of the conjunctiva is smooth except over the caruncle and at the tarsal edge where it becomes slightly mucous. On the plica semilunaris are small nodules and at the canthi it becomes somewhat velvety.

The conjunctiva may be inflamed in various degrees and may conform to various types which have certain symptoms in common, namely, photophobia, which is commonly, but not invariably present; blepharospasm, occasionally present; increased secretion; hyperemia, with swelling of the papillæ giving rise to a velvety appearance of the conjunctiva; and in some instances a false membrane formation.

Conjunctivitis is the generic name given to an entire group of diseases. A classification according to the bacteriologic findings has many advantages, being more scientific and exact than the older nomenclature. The disadvantages of this classification is that there are many organisms normally present in healthy conjunctiva which may, however, become pathogenic when the resistance of the conjunctiva is lowered by mechanical or chemical irritants.

The delicate conjunctiva of the newborn is subjected to insult from the moment the journey begins from the security of the mother's uterus. Even that citadel of safety is not invariably inviolate because numerous instances are on record where infants have been born suffering from an undoubted purulent ophthalmia, usually of gonorrheal origin. Children have been born with perforated corneæ and eviscerated globes from intra-uterine conjunctivitis. This may readily be comprehended, granting the incidence of early ruptured membranes, a brow or face presentation and a very slow delivery, where there is every chance of the eyelids being rubbed open and the conjunctiva exposed to contact with an infected vaginal mucosa. The injudicious examining finger may directly injure or infect the conjunctiva. A case has been reported by Druais of a child delivered by Cesarean section in which the eyes were red and swollen, and bathed in pus at the moment the head presented through the membranes. The infection was shown to have been due to streptococci.

After running the gauntlet of all these dangers from infection the infants eyes are exposed to irritants, either very rightly instilled as a prophylactic measure, or accidently entering the eyes while bathing, or from the fingers of attendants or relatives, and even through the medium of dust ladened air and by flies or others insects.

In the majority of the cases at birth and for one or two days thereafter the conjunctiva are free from bacteria, and have been found free of bacteria up to the sixth day. Contamination usually takes place early, first with the Xerosis bacillus and the staphylococcus albus. Parsons reports a small diplococcus probably of mixed species easily mistaken for the pneumococcus as a frequent habitat of the conjunctiva. The pneumococcus is present in about 4 per cent., occasionally staphylococcus pyogenes aureus, rarely streptococcus pyogenes and the influenza bacillus. The natural immunity of the newborn to infection is very great, but conjunctivitis is estimated to constitute about 30 per cent. of all ocular diseases in the adult and constitutes a much larger per cent. in the newborn.

Gonorrheal Conjunctivitis (Ophthalmia Neonatorum).—Prophylactic measures instituted at birth have so reduced the incidence of gonorrheal conjunctivitis that it is now rarely seen in private or public hospitals, when a rigid prophylactic regime is insisted upon. It is considered rather a stigma for such a case to occur in an institution and its infrequency may lull us into a feeling of false security. We should not forget also that there are cases of virulent purulent conjunctivitis caused by other infective agents than the gonococcus, which may even proceed to destruction of one or both eyes. For instance, staphylococcus, streptococcus, pneumococcus, pyocyaneus albus and colon bacillus. Druais asserts that only one-half of ophthalmia neonatorum purulentia is due to the gonococcus. This, it must be remembered, might have a very important medicolegal bearing.

The necessity of prophylactic instillations is universally conceded, but some disagreement has arisen as to the best reagent to use. It does not seem to make a great deal of difference whether the classical Credé 2 per cent. solution of silver nitrate, or a weaker solution, is used or whether one of the organic preparations, such as, Argyrol in a 10 to 20 per cent. solution, Protargol, Silvol, or Neosilvol is employed. A silver nitrate solution of a strength of from 1 to 2 per cent. when fresh is the most reliable. The great objection to it is the possibility of irritation. It should be emphasized that nurses and all attendants who have the care of newborn children should be instructed to handle the delicate eyelids of babies with the utmost gentleness, and to take the greatest precautions, not only to do no mechanical injury by rough handling, but also not to cause hyperemia or possible exfoliation of the epithelium by allowing extraneous irritating matter such as soap to enter the eyes. This latter is a frequent source of the mild but tedious and annoying inflammations of the conjunctiva in babies.

Every inflamed eyelid in the newborn, whether or not accompanied by a purulent discharge, should be given immediate attention. Diminishing prevalence of gonorrheal conjunctivitis in the newborn must not beget carelessness or indifference to the ever present danger. There is no reason to believe that there is less gonorrheal vaginitis than formerly, although, the virulence of the infection may have diminished. The 1918 report of the National Committee for the Protection of Blindness gives 23 per cent. as the proportion of the blind from this cause.

With a sterile platinum loop a bit of discharge from the lower fornix should be taken and a smear made. A culture should be made at the same time since it has been repeatedly shown that smear examinations, in from 30 to 50 per cent. of cases, may fail to show organisms demonstrable in culture. If a Gram-negative intracellular diplococcus is found, treatment should be instituted at once without waiting for confirmation by culture.

Incubation.—Conjunctivitis neonatorum may begin as early as 12 to 48 hours after inoculation. Those cases occurring before the second day after birth are probably due to intra-uterine infection, and those occurring after 10 days to secondary infection. The average time is three days. When soiled fingers, cloths, sponges, etc., are the infecting agents the delay may be greater.

Credé explains some late infections by assuming that the organisms may remain dormant within the Meibomian glands and reach the conjunctiva later.

The eyes are most commonly attacked alternately, one preceding the other by a short interval. Treatment should be continuous night and day, interfering as little as possible with the infant's sleep and feeding.

If only one eye is affected, as occurs in one-half the cases, the other eye should be protected with a glass shield. Cold compresses composed of pieces of gauze 2 in. square and several layers thick, may be applied every two minutes for 15 minutes at a time. In infants the procedure cannot be kept up continuously because of the danger to the nutrition of the cornea. There should be an interval of 15 minutes to one-half hour between applications.

The eyes must be kept clear of pus by irrigations of tepid boric solution every half hour. Argyrol in a 25 per cent. solution instilled every 15 minutes has proved very efficacious, as has a 10 per cent. solution of protargol in glycerin instilled three times daily. The great danger is to the cornea, and attendants must be cautioned to use the greatest care not to abrade it. If the lids become so swollen and thick as to mechanically endanger the cornea a canthoplasty should be done. The use of steam, while apparently followed by brilliant results in adults, is impractical in infants.

Injections of cow's milk, boiled four minutes, may be frought with danger unless cautiously used. The careful and intelligent use of this routine, always emphasizing the imperative need of great caution in all manipulations not to abrade the cornea, will usually forestall perforation of the latter. The inflammation of the conjunctiva is self-limiting even without medication. But it is the involvement of the cornea which constitutes the great danger. There is no doubt that the gonococcus has the power to invade the corneal epithelium without previous denudation, but this greatly facilitates its invasion.

The prognosis depends on the violence of the original infection, the intensity of the inflammation, and the intelligence and the conscientiousness of the treatment.

Idiopathic Conjunctivitis of the Newborn.—There is a type of inflammation of the conjunctiva of newborn children occurring from 5 to 10 days after birth, characterized by some edema of the lids, hyperemia, slight purulent discharge, photophobia and blepharospasm. The smear and cultures are usually negative, or only such organisms are found as are usually present in the conjunctival sac.

Such inflammations are often a mechanical or chemical conjunctivitis caused by manipulations of the lids or by irritating substances entering the eyes. When an infectious origin can be excluded a masterly inactivity is indicated. Beyond cleansing the eyes free of secretions, with boric acid or normal saline solutions, nothing should be done.

Another type shows an actual structural change in the conjunctiva, a folliculosis possibly. These are best treated by an occasional light brushing with a 0.5 per cent. or 1 per cent. silver nitrate solution.

When a mild infection is present mercurochrome in a 1 or 2 per cent. solution may be used, a drop, two or three times a day, or a 0.25 per cent. zinc sulphate solution. It must always be borne in mind that it is very easy to overtreat infants' conjunctivæ, and their natural resistance to manipulations about the eyes makes the eversion of the lids difficult. There is danger of doing damage by rough and unskillful handling, hence, the ever present necessity of using all gentleness in their care.

ICTERUS NEONATORUM

Icterus neonatorum appears as a yellowish discoloration of the skin and sclera which occurs to a greater or less degree in a large percentage of children during the first few days of life and is usually a physiologic process.

Etiology.—Much has been written concerning the cause of this form of jaundice of the newly born and many different theories have been advanced to account for it, the most accredited of which are, first, that the jaundice is due to a large amount of bile in the blood of the newborn, and second, that it is due to an excess of coloring matter in the blood from a rapid breaking down of the red-blood cells immediately after birth. It is now conceded by most investigators that the first theory is the correct one. Ylppo found that the blood of the icteric newborn contained no greater amount of coloring matter than was to be found in the blood of the non-icteric. He believes that the jaundice appears because the liver after birth allows a part of the bile coloring matter to pass into the blood, just as the fetal liver did. Bang found that the blood in the umbilical cord always contained a notable amount of bile pigment and that all children examined showed a constant increase in bile pigment in the blood during the first few days after birth. Hess believes that icterus neonatorum is the result of a defective correlation of the excretion and secretion of bile, the rudimentary excretory organs being unable to cope with the sudden profuse secretion of bile. In most cases of marked jaundice he found an excessive excretion of bile into the duodenum and stomach. Quincke's theory that the jaundice is due to the patency of the ductus venosus is often quoted. According to this theory the bile passes directly from the intestine into the portal vein circumventing the liver and entering the inferior vena cava. This duct is often found however, to be open as late as the fourth week of life and after all jaundice has cleared up.

The hematogenous theory has been strongly supported and cannot be dismissed without discussion. Heiman in 1912 brought forth proof to show that there is an actual destruction of red-blood cells directly after birth, thus refuting the argument that the apparent disappearance of red cells is due to an increase in blood plasma, causing an apparent and not an actual decrease in the number of cells in the blood stream. This destruction of the red cells causes a liberation of hemoglobin which is taken up by the liver and transformed into bile pigments. It would seem, therefore, that the two theories may be closely related and possibly after all the excess bile secretion at this period may be partially due to the destruction of erythrocytes.

Pathology.—Little if any pathology is found in this condition. Most of the tissues of the body are more or less jaundiced. The liver presents only small areas of jaundice and the spleen and kidneys none at all. Bilirubin crystals are frequently found in the kidneys.

Symptoms.—Simple icterus neonatorum occurs in from 60 to 80 per cent. of all the newborn. It usually appears between the second and the fifth day and, depending upon the degree of discoloration, lasts from 3 to 10 days but may continue in severe cases as long as three weeks. Usually all parts of the body are equally jaundiced, except that in the mild cases, the sclera shows no discoloration. When it does become jaundiced it is apt to be the last area involved and the last to return to normal. The mucous membranes of the mouth are affected in most cases. There is no discoloration of the feces and ordinary tests will disclose no bile in the urine although very delicate tests may show a trace of bilirubin. There seems to be no constitutional embarrassment, pulse and respiration rate remaining normal. Breast milk is digested normally, no fever appears and the weight curve is uninfluenced by the degree of jaundice. The tendency toward hemorrhagic disease is not increased in this form of icterus. While the condition may appear in any type of newborn, the premature and the under-nourished infants are most subject to it.

Diagnosis.—There are several forms of jaundice which must be distinguished from simple icterus neonatorum. Congenital malformation of the larger bile ducts causing obliteration or atresia of the lumen is not extremely rare and must be differentiated from ordinary icterus. The jaundice in these cases may be present at birth and has been so reported in a number of cases. However, the point of differentiation would be made on the progressively increasing jaundice with an absence of the normal coloring of the stool, which will be very pale or white, and also on the definite increase in the size of the liver and spleen. Another not uncommon form of jaundice in the newborn is that which accompanies sepsis. This, however, does not appear until the fifth or sixth day and is accompanied by fever, loss of weight, and possibly other signs of septic process, such as infection of the umbilicus, peritonitis, or pneumonia. Congenital syphilis is often accompanied by jaundice but it is apt to appear later than in simple icterus neonatorum and hemorrhages are frequent. The usual signs of congenital syphilis would probably be present to assist to a correct diagnosis.

Prognosis.—This form of icterus is usually unassociated with any disturbance of a normal function and the ordinary progress of the child is uninfluenced.

Treatment.—Icterus neonatorum presents no problem of treatment, as the jaundice clears up in a few days without therapeutic interference.

DISEASES OF THE SKIN

Introduction

A larger proportion of skin affections are seen during the first weeks of life than at any subsequent period. Comparatively few infants entirely escape some form of the dermatoses. Several factors combine to produce a special tendency toward these affections. At birth the integument is covered by vernix caseosa, a cheesy deposit, secreted by the sebaceous glands of the fetus itself, along with exfoliated epithelium. With its removal the soft, tender, hypersensitive skin, with its thin epidermis and a corium of comparatively slight resistance, is left exposed to various irritants. The epithelial cells of the skin of the newly born are less coherent than in later life, and slow desquamation continues to take place. At this time the sebaceous glands are developed to a greater extent than the sweat glands, perspiration being seldom seen in a healthy infant during the early weeks of life. The functional activity of the sebaceous glands and overaccumulation of oil has an irritating effect on the skin and often causes seborrheic masses to collect, particularly on the scalp. Excessive washing or rubbing to remove this oily condition is apt to leave a dry thin epidermis deprived of the normal amount of sebaceous secretion necessary for its protection.

On the other hand, cleanliness to insure against irritation from decomposition on the skin of mucus or milk about the mouth, or of the urine or feces is of course essential. In cases of auto-intoxication, through faulty metabolism, toxins in the intestinal canal may be resorbed into the circulation and result in urticarial manifestations; or it may be possible that the microörganisms themselves are so carried, such an origin having been suggested in cases of erythema exudativum. Atrophic, malnourished infants have noticeably slight resistance to skin affections, as well as the nursing baby which is overfed, in which cushions of excessive fat inhibit the circulation to the skin, resulting often in eczema. The hypothesis that young protoplasm is very irritable, may explain the severity of the lesion often resulting from apparently slight causes. In the treatment of these conditions the causative factor should of course be determined whenever possible and removed. Only the most non-irritating and soothing medication should be used locally and the parts affected should be protected from every possible further irritation. Only the most recent lesions should be sought out for diagnostic purposes, as a previous application of drugs may have so changed the appearance of the eruption as to obscure the original clinical picture.

ERYSIPELAS

Erysipelas, always a severe infection, is particularly virulent when attacking the newborn. The general characteristics of the infection in infants are the same as in older children, but the feeble resistance of the newborn to this form of infection allows the disease to spread rapidly, and in the large majority of cases causes fatality, the mortality rate being given as high as 95 per cent. Erysipelas of the newborn rarely occurs in a well-conducted institution, the disease usually arising from infection of the umbilical wound, or an abrasion of the skin. When first appearing around the umbilicus, the disease is seldom uncomplicated, a phlebitis, and often a peritonitis being present as a rule. Erysipelas is generally supposed to be caused by a specific streptococcus, but it is doubtful if this is always true in the case of the newborn as in them it is apt to be associated with other pyogenic infections. This leads to the surmise that some other active pyogenic organism may be responsible for the erysipelas.

Symptomatology.—The onset is usually sudden with a sharp rise in temperature, and the appearance of the characteristic inflammation of the skin. There is great prostration, the infant nurses but little, and is apparently toxic. The inflamed area spreads with great rapidity. In some cases huge blebs form when the disease arises at the umbilicus, and it may cover the entire abdomen within 48 hours. When the face or extremities are involved, edema and swelling are pronounced. Death frequently occurs from the bronchopneumonia or peritonitis which so often is a complication.

Treatment.—Isolation is of the utmost importance. Every effort should be made to maintain the nutrition, and control the temperature, other general treatment being of little avail, except the control of untoward symptoms as they arise. Locally, wet compresses of saturated solution of magnesium sulphate, continuously applied over the affected area, are of great value. The local application of ichthyol ointment, 10 to 25 per cent. strength, is highly recommended. This ointment, the base of which may be petrolatum, or equal parts of lanolin and cold cream, should be spread upon a cloth and applied several times daily as a plaster.

Sclerema Neonatorum

Usembezius, in 1718, made the first report of a case of Sclerema Neonatorum, but to Underwood is credited the first precise description of the disease in 1784. Clementowsky in 1873 and Parrott in 1877 added valuable contributions to our knowledge of the subject. There is much confusion in the literature as regards edema neonatorum, sclerema neonatorum, and scleroderma neonatorum. This confusion has given rise to the multiplicy of names given to these diseases by various writers. A few of those that were recognized for a time are Underwood's Disease, Scleroderma Neonatorum, Sclerene des Enfants, Oedematie Concrete, Thirial's Disease, L'endurcissement Athrepsique, Sclerom der Neugeborenen, Hidebound Disease, Scleroma, Sclerisma, Sclerema, Oedematia, and Scleroedema. The extreme rarity of the disease now known as Sclerema Neonatorum, and the fact that it occurs very early in life, and usually proves fatal within a few days, has much to do with our lack of accurate description and classification of it. The clinical picture published by Parrott in 1877 is generally accepted as classical, he being the first writer to point out that sclerema neonatorum and edema neonatorum are distinct and separate conditions. While Billard gave an excellent description of edema neonatorum, he did not believe that true sclerema existed until after death, or at the earliest in infants actually at the point of death. Clementowsky, in 1873, recognized three varieties of sclerema, namely, the erysipelatous, the edematous, and the adipose. The latest interpretation of this condition, given by Patterson and others, is that sclerema neonatorum occurs in different degrees of severity. It may be so slight as to be confused with sclerederma or so-called pseudosclerema or so intense as to be complicated by an edematous condition, which accounts for many authors confusing it with edema neonatorum, or it may appear in the classic form which is usually referred to as sclerema adiposum.

Etiology.—Sclerema is not necessarily associated with or secondary to any other recognizable morbid condition and the etiology is mainly speculative. Leger, in 1823, suggested that one of the main factors was the effect of cold, causing coagulation in the subcutaneous tissues, and a favorite idea of many writers of today is that this change occurs because the fat of the newborn contains a greater amount of stearic and palmitic acids and considerably less oleic acid than that of the adult. The theory of nervous influence was first brought out in 1818 by Liberali and has been adopted with modification by later writers, particularly Money in 1899 and Somma in 1892. Somma's ideas are based on the theory of thermogenetic, thermolytic and thermotactic centers in the brain. He holds that the chilling of the surface of an infant suffering from congenital debility would be sufficient to upset the regulation of these centers and cause the local changes in subcutaneous tissue. A disorder of the internal glandular secretions has also been suggested as a possible hypothesis, the affection being manifested in conditions somewhat similar to those found in myxedema. The more modern explanation and the one which lends us the most hope is that of bacterial invasion. Eustice Smith, Jemma, and Comba have devoted much time to this theory, but as yet have obtained no results that would demonstrate the disease as resulting from the action of any specific organism. It is thought that the toxemia from some bacterial infection would most readily explain the multiplicity of conditions which are found.

Prematurity, syphilis, diarrheal diseases, and general atrophy appear to be predisposing causes. Atelectasis is frequently noted in connection with the disorder, but heredity appears to play no part.

Pathology.—Patterson gives a comprehensive description of the pathology of the disorder. He states: "The skin and subcutaneous tissues are dry and hard, there is extreme atrophy with consolidation of the skin, including the rete Malpighii, of which the cells are scarcely visible, forming a compact mass, with ill-defined contours. The horny layer is unchanged. In the subcutaneous fat the fibers of the connective tissue are more numerous than usual and thicker, and the fat itself is considerably diminished. The fat cells are nearly entirely deprived of their fat but their nuclei can be distinctly seen. The blood-vessels, especially those of the papillæ of the skin, are narrowed to such an extent that one cannot distinguish their lumen."

Symptoms.—The disease may be present at birth or occur within the first 10 days, or infrequently it may develop later, within the first few months. It usually manifests itself first upon the lower extremities and more or less rapidly invades other parts, the general involvement becoming complete within three or four days. Every part of the body surface may become affected except the palms, soles, scrotum, and chest. The distribution is symmetrical and the affected parts have the appearance of atrophy rather than swelling. The skin appears stretched, its surface being remarkably smooth. It looses all flexibility and is firmly united with the subcutaneous structures. It becomes absolutely impossible to separate it from the adjacent parts and as the tension and hardness daily increase, it acquires the feeling of thick leather. As Parrott says, "It appears as if all the soft parts were coagulated." The integument is not pitted by pressure as in an edematous condition. The limbs become immobile and but for the movements of the thorax and face the body seems to be in a state of cadaveric rigidity, or it may suggest having been carved from wood, percussion over the affected parts giving a board-like note. The color of the skin has been described as either pale, yellowish, or waxy at first, later becoming faintly bluish-red or livid, and it is cold to the touch. The process may become so extensive and involve the face to such a degree that it is impossible for the infant to open the mouth. The temperature falls soon after birth and seldom returns to normal. A temperature as low as 95 degrees is common though it may fall to 85 degrees, and cases are reported where it has been as low as 72 degrees. There is marked loss of weight. The fontanelle becomes depressed, the mucous membranes are dry, respiration is superficial and slow, often less than 18. The pulse is slow, frequently 60 or even less in some cases and, because of the skin condition, difficult to find. There is usually cyanosis, and atelectasis has been frequently noted. Many of the infants are jaundiced, and hemorrhages into the lungs and heart have been noted at necropsy. Diarrheal conditions often prevail if death does not occur in the early stages.

Diagnosis.—The confusion in the minds of many authors as to the exact nature of this disease makes it very difficult to differentiate this condition from what may well be considered allied diseases. Some authors describe as a distinct affection a condition which they name sclerema edematosum or edema neonatorum, which in every way corresponds to what other contributors believe to be an ordinary sclerema adiposum accompanied by some degree of edema. Still others describe edema neonatorum as merely a sign of a disease rather than as a disease itself, it being commonly secondary to some derangement of the lungs, kidneys, or vasuclar system. In differentiating edema from sclerema one must remember that in sclerema we do not have the characteristic urinary findings, and also that there is no pitting on pressure, and that the eyelids, scrotum and labia majora are not involved. Erysipelas neonatorum ordinarily presents definite areas of acute inflammation of the skin with sharply defined and advancing margins, and fever is usually present.

Prognosis.—Sclerema may present varying degrees of severity and is usually not secondary to any other disease. The prognosis, while commonly bad, varies directly with the extent of involvement and the severity of the constitutional symptoms. Those conditions described as pseudosclerema, are thought by Patterson to be only mild cases of true sclerema from which the patient normally recovers. In the graver form the infant usually dies within a few days.

Treatment.—Every attempt should be made to preserve the body heat, a very excellent method being to place the child in a "premature basket" with the customary hot-water bottles. An incubator may be used if available, but has no advantages over the basket. Breast milk should be fed either by bottle or gavage without removing the patient from the basket. Gentle rubbing of the body and limbs with warm olive oil may be of benefit. Cardiac stimulants are indicated to support a failing circulation. Wassermann tests should be made in all cases and also blood cultures in the hope of isolating some specific organism which might be combated by vaccine therapy.

Congenital Defects and Scars

Congenital skin defects of the newly born are infrequently mentioned in the literature. Abt has collected some 36 case reports.

Campbell, Billard, and Dieterich were among the first to report cases of this condition. All of these were defects of the scalp. Mace, in 1901, Muller, Burger, and Emanuel, a little later, have reported defects and scars on other portions of the body than the head, but with these exceptions nearly all cases reported have been of the scalp.

Etiology.—These defects and scars have been attributed to various causes, prominent among which are errors in development and inflammatory adhesions between the external layers of the skin and the amnion. According to Abt, it is the tearing away of these adhesions, the so-called Simonart's bands, from the fetal skin which leaves the congenital defect. It has been noted that such defects often are associated with other congenital malformations. Trauma to the uterine wall is often mentioned as a cause of skin defects in the newborn.

Pathology.—Lesions occur most frequently on the scalp. However, they may occur on the body or limbs. In Abt's case there was a bilateral skin defect of both knees. In Burger's case, the lesion on the scalp was accompanied by lesions on both knees and elbows. In Emanuel's case there were defects on hands, legs, feet, over the temples and on the bridge of the nose, also in the middle of the lumbar region. Lesions are usually visible at birth and undoubtedly originate *in utero*. They present ulcerated areas which affect both layers of the skin. The subcutaneous tissue usually is not involved. Keller found that minute hairs occurred in the affected area, but this does not conform with reports by other authors. Microscopically, a failure of the epidermis to develop is shown and the skin glands are rudimentary, if developed at all. There are no signs of old or recent degeneration, which would tend to exclude these defects from being a secondary process, due to some intra-uterine inflammation. The absence of epithelial structures, smooth muscle and adipose tissue, and the well-developed connective tissue, seen microscopically, would indicate that the skin defect may be traced to an early period of embryonal life. The very early development of these lesions would argue against their ever being due to injury at the time of birth.

Prognosis.—Unless the skin defect is wide in extent there is no apparent reason why the slight malformation should, of itself, affect, unfavorably, the life or general health of the infant.

Treatment.—As there is no inflammatory reaction or degenerative process in the region of the defects, no special treatment is required. The majority of these lesions tend to cicatrize in a short time, thus providing an adequate covering for the denuded area.

Miliaria

Miliaria is described as a mild inflammatory infection of the skin characterized by discrete pin-point or pin-head sized papules and vesicles occurring at the mouths of the sweat ducts.

Etiology and Pathology.—Much discussion has taken place regarding the etiology and pathology of this condition. It is very commonly found on the skin of the newborn but is usually of so mild a character as to excite little or no attention. It is generally conceded that very slight if any functional activity of the sweat glands is present during the newborn period, which would argue against the theory that the affection is due to sweat obstruction. From the generalized and irregular distribution of the lesions one would conclude that they were of purely inflammatory origin, not intimately associated with the function of the sweat glands but produced by friction of various irritants such as soaps, oils, or clothing on the hypersensitive skin, thus opening the gland ducts to low-grade infections. The lesions as seen are usually papular but many vesicles may be present these having a faint red areola surrounding the yellowish-white center. Occasionally the contents become seropurulent or even purulent. Very occasionally in the newborn, a typical area of that variety of miliaria known as sudamina is found. These lesions are principally located on the back and the neck. They form rapidly, do not increase in size, are closely set together, and do not coalesce. The contents are at first clear but later become opaque with but little inflammatory reaction.

Symptoms.—This condition gives rise to no general symptoms. The temperature is not disturbed and the infant usually shows no sign of discomfort.

Prognosis and Treatment.—The affection is common in the newborn and is very transitory in character, lasting from a few hours to four or five days and disappearing spontaneously without treatment. Occasionally, a few of the larger lesions will show evidence of actual bacterial invasion, forming a tiny abscess or furuncle which should be opened and touched with alcohol or silver nitrate. Iodin must be used very cautiously on the skin of infants as it is very likely to set up a severe dermatitis.

URTICARIA

Urticarial manifestations are very often seen during the first few days of life, as large red slightly raised blotches on the body and extremities. They have been noted by the writer as early as the beginning of the third day, before any food has been taken. The lesions are quite transitory in character, disappearing usually within 24 hours, and accompanied by no constitutional symptoms. The disease undoubtedly arises from some very slight cause which as yet is problematical, possibly from the absorption of toxins from the intestinal canal.

TELANGIECTASIS

Telangiectasis in the newborn usually takes the form known as nevus araneus or spider nevus. It is the simplest form of vascular nevus and may be congenital in origin, or may appear after birth.

Etiology.—No definite cause for the appearance of these nevi of the newborn has been determined.

Pathology.—These nevi consist of a dilated central capillary with a number of smaller capillaries radiating from it.

Symptoms.—This nevus is most often found on the face of the newborn in the region of the nose. It is usually single but occasionally several are present. Most often the surface is level with the skin, with the central capillary about the size of a pin head forming a red dot from which radiate in either a straight or tortuous manner thread-like red lines usually not exceeding ¼ in. in length. These nevi may be scarcely noticeable at birth, and rarely attain conspicuous dimensions. Their tendency is toward spontaneous disappearance, and aside from being a slight disfigurement, they are of no clinical importance.

Treatment.—Treatment of this condition is seldom if ever necessary during the newborn period. If for any reason it is deemed best to remove the nevus, a cure may be secured by the destruction of the central capillary either by simple puncture or by introducing the electric needle into the red dot.

Scleroderma in the Newborn

(Hide-bound Disease; Scleriasis; Dermatosclerosis)

This is a rare affection of the skin characterized in general by an infiltration of the skin substance, followed by shrinking or contraction, and by impairment of the secreting apparatus, resulting in a stiffening and hardening of the integument. It occurs in diffuse and symmetric forms which spread more or less rapidly over the surface of the body. The affected area may be pigmented or have a lardaceous appearance with pinkish borders. At the onset it is especially likely to involve the chest, face, neck, scalp and hands.

It is questionable whether this condition should be described as a disease of the newborn, as it is rarely authentically reported as such. In the old literature much confusion in terminology is found and the name has been used interchangeably with that of sclerema. Forget, in 1847, and Gillette, in 1854, insisted that these two conditions were separate and distinct, but Roger, Le Breton and Hennig, as late as 1877, held to the view that the two conditions were identical, maintaining that the pathologic difference was due only to the variation of structure of the tissues at the different ages. However, it is being gradually accepted that the two conditions are in no way related. For many years scleroderma was known as a disease of the adult only (scleroderma adultorum), but over 100 cases in children have now been reported in the literature, a few cases in the newborn. In 1876 Cruse reported what he believed to be four cases of scleroderma in the newborn, but Cockayne,* analyzing them in 1916 believed that they were all cases of sclerema. Neumann, Langmead, Barker, and others have all reported cases. Lieberthal† (1918) states that many cases recorded under the title of sclerema of the newborn are really cases of true scleroderma and among these mentions those of Money, Bunch, Blacker, Carpenter and Parkinson, Welt-Makels, Sobel, Myers-Wright, Monad, Barr and Browning. Cockayne believes that some cases described as of congenital scleroderma are undoubtedly instances of annular or semi-annular fibrous constrictions, probably caused by the pressure of amniotic bands or coils of the umbilical cord.

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* Brit. Jour. Dis. Child., August, 1916.

† Jour. Cutan. Dis., January, 1918.

Etiology.—The most satisfactory explanation is that this condition is a trophoneurosis due to a disease of the sympathetic nervous system. The distribution of the skin lesions often corresponds to segmental areas, spinal areas, or to the course of cutaneous nerves. In adults, scleroderma and disease of the ductless glands often affect the same individual. Myxedema, exophthalmic goiter and Addison's disease have been found coincident with scleroderma. Cockayne suggests that the diseases of the skin and the ductless glands are probably due to a disorder of innervation.

Symptoms.—The symptoms of scleroderma in the newborn appear to differ very little if any from those occurring in older children and adults. The children, as described in the literature, have been well developed with no derangement in the temperature, the symptoms being entirely local. As a rule, the lesions first occur on the face, chest, and upper extremities, and consist of isolated and irregularly distributed solidified plaques and nodes which may soon coalesce. The skin is firm at first, later becoming hard and board-like and the surface dry and scaly.

Diagnosis.—Sclerema is the disease most likely to be confused with scleroderma. The normal temperature and the distribution of the lesions, on the face and neck, however, present a clinical picture exactly the opposite of sclerema in which the temperature is subnormal and the back and buttocks are most commonly and most severely affected. It must also be considered that the mortality in sclerema is very high but in the event of recovery the skin condition will clear up within a few weeks with seldom if ever permanent atrophy of the skin. Care should be taken not to mistake the skin defects and fibrous constrictions due to amniotic adhesions and bands, or pressure from coils of the umbilical cord for isolated patches of scleroderma.

Treatment.—Every effort should be made to maintain the nutrition of the patient. Local applications of oils or ointments and gentle massage are indicated. Thyroid gland has been given in many cases but has not proved beneficial.

Pemphigus Neonatorum

(Pemphigus neonatorum contagiosus; Pemphigus epidemicus)

Pemphigus neonatorum is a contagious disease of the skin usually epidemic among the newborn. It is characterized by the formation of a few or numerous irregularly scattered and variously sized, rounded or oval blebs filled with clear fluid, arising from a slight papule or from apparently normal skin. The disease may be clinically benign or malignant depending on the degree of constitutional involvement.

Etiology.—The disease was described as early as 1610 by Forastus and again in 1773 by Kraus who stated at that time that he had repeatedly seen epidemics of pemphigus among newly born children. The disease has been frequently described as occurring throughout the civilized world both endemically and epidemically. The epidemics have nearly all been mild or benign in type, the more malignant form occurring less often. Most authors consider the term "pemphigus" unfortunate as applied to this disorder in the newborn as the condition is in no way analogous to the affection known by the same title in adults. Pemphigus occurs in all races but seems to be most severe in children of the white race born in the tropics. It is more often seen in cities than in the country districts and is most apt to occur in hospitals where large numbers of the newborn are cared for. When the disease appears, it occurs with equal frequency in any of the children regardless of size or condition of nourishment, and the incidence is about the same in both sexes.

Some strain of the staphylococcus pyogenes aureus is generally conceded to be the offending organism although streptococci have been found in the lesions occasionally, especially in those of the malignant type. Some investigators have described the organism as belonging to the diplococcus strain while others advance the theory that pemphigus neonatorum is analogous to impetigo contagiosus of older children and adults. This theory is supported by the fact that mothers who are nursing babies afflicted with acute pemphigus frequently develop lesions on the breasts that appear to be typical impetigo. It has also been noted that an infant in which the blebs on the body are typical will develop lesions on the scalp that are of impetiginous appearance. Von Reusse states that the disease is both ectogenous and endogenous, and that the endogenous variety is apt to be malignant. The method of invasion is not known although the disease is undoubtedly spread by direct contact. A certain percentage of the children will show first signs of the disease on or about the fifth day after birth, probably representing an incubation period of from three to five days, assuming that they were exposed directly after birth. Some children seem to be immune, or they escape exposure, as they show no evidence of the infection, while others do not develop the first signs of the disorder until as late as the beginning of the third week. These cases probably escape the original exposure but contract the disease from other developed cases. Intimate contact does not seem to be necessary and it is believed that the infection is transmitted by medical attendants, nurses, midwives, and by the various materials used in caring for the newly born. In hospitals in which it has been prevalent, it may entirely disappear for a period of a week or more only to recur as extensively as before. Trauma to the skin of the infant, which has been thought by some authors to favor the pemphigus invasion, evidently plays a small role, although the first lesions are very apt to occur in the region of the axilla where pressure from the nurse's hands in lifting the baby is likely to be greatest. The idea that the original infection may occur in the parturient canal has been advanced, but there is little to support this theory.

Pathology.—The pathology of this disease as described by Sabauraud in 1900 is briefly as follows: The disease is divided into three stages, the prevesicular, the vesicular and the postvesicular. In the first stage a very thin superficial epidermal layer appears, covering a deeper layer of flattened cells with intercellular edematous spaces, and the infiltration of the leukocytes into serous spaces of various sizes. No organisms are found. The vesicular stage is characterized by five main features: First, the thinness of the horny layer of epidermis forming the cover of the vesicle. This never raises any of the underlying tissue with it. Second, the small number of formed elements in the early stage. Third, the relatively large amount of serum which is clear at first and later becomes crowded with leukocytes. Fourth, epidermal and dermal edema due to a serous intercellular exudate; perivascular leukocytic infiltration of the derma; thin bands of leukocytes in the spaces of the epidermis at the stratum lucidum close to the floor of the vesicle. Fifth, organisms can be found when the vesicle is fully developed. In the postvesicular stage a crust appears composed of a thin horny layer of coagulated serum with enmeshed leukocytic nuclei. The bacteriology of the crust is variable, staphylococci predominating.

In malignant cases streptococci and staphylococci have been found in blood cultures. No gross pathology has been discovered in the fatal cases, and it is suggested that death may be due to the widespread destruction of the skin as in cases of severe burns. However, the absorption of toxins from the extensive skin lesions probably plays no small part in the fatal termination.

Symptomatology.—Clinically pemphigus neonatorum occurs in benign and malignant types. The picture of a well-developed case of pemphigus is characteristic. Usually between the third and the eighth day there is a sudden appearance of vesicles or blebs resembling coarse cysts. The axilla, groins and neck are the favorite sites of onset. The first symptom is a reddish appearing macule. These grow rapidly and a thin pellicle forms over the spot from which the vesicles or blebs develop. These are filled with a fluid which is at first clear but soon becomes cloudy. The vesicles at first may be very small and tense but they grow very rapidly, spreading in the periphery under the thin horny layer of the epidermis which forms the cover of the vesicle. The lesion becomes flabby and shows a tendency to coalesce with neighboring blebs. They may reach the size of a hazel nut or even larger and may spread to cover any part of the body, although the palms of the hands and the soles of the feet are usually exempt. The thumbs and fingers however are often affected. Whenever a vesicle develops in a fold of the skin as in the axilla, neck or groin it is almost sure to infect the skin of the opposing side of the fold, developing a new bleb. The lesions are likely to burst before reaching maturity, but they will continue to spread in the periphery. The fluid escaping from the lesion may be scanty or abundant, and has a yellowish serous appearance in the ordinary case. The discharged vesicle leaves a red, moist, exudating surface with a margin of epidermis, the affected spots remaining hyperemic for some time. In the malignant type the exudate may be of a dark greyish purulent character. New lesions are apt to appear from hour to hour and while in many mild cases the original site is the only area involved, and the whole process may even be limited to two or three vesicles, the disease is much more likely to spread to other parts of the body, often envolving the limbs, trunk, face and scalp. In a well-developed case, the duration of which is from three to five weeks, lesions will be found in all stages of development at once. In the benign type there is no indication that the vesicles cause any pain or itching, and the baby is apparently comfortable. As a rule, the normal gain in weight continues undisturbed and the temperature remains normal throughout the illness. In the malignant type the vesicles develop with an astonishing rapidity and quickly involve large areas of the body, which are soon denuded from the destruction of the epidermal layer. A temperature of 104 degrees or more is common and the child has all the appearance of a fulminating septic infection which usually rapidly proves fatal. It is in these cases that a positive blood culture may be expected.

Prognosis.—The prognosis in this condition depends very largely on the type of the disease and the amount of constitutional involvement. In epidemics the type is nearly always benign. The child's general progress is undisturbed by the skin affection and complete recovery takes place in from three to six weeks. The lesions, even though extensive, heal without leaving any scar, although some areas may be slightly hyperemic for some weeks longer. In these epidemics a fatality seldom occurs. Even the prematures of low vitality will survive a rather extensive involvement of the skin. In the fulminating or malignant type, more than 50 per cent. of the cases prove fatal, many of them within a few days after the onset.

Treatment.—When once this disease invades the nursery of the newborn, it is most difficult to eradicate. In certain institutions, where great care is not exercised and proper technique carried out almost every baby born contracts the disease. When a case of this disorder develops no pains should be spared to secure the most rigid isolation of the patient, and the newborn ward and delivery room should immediately receive the most thorough cleansing possible. Nurses and attendants should be carefully examined for any skin lesions and if any are found that even remotely resemble impetigo, they should be excluded from contact with both mothers and babies. If possible all newborn babes who were in any way exposed to the infected case should be treated as potential cases and should not be returned to the renovated nursery. The infected child should be cared for by attendants and nurses who in no manner come in contact with others of the newborn or with the mothers. Nurses should be advised of the highly contagious nature of the disease and should wear rubber gloves or use antiseptic washes such as 1 to 2000 bichlorid of mercury solution when handling the baby. In the treatment of the benign cases the greatest effort should be directed against the spreading of the lesions. This sometimes seems to happen in spite of every precaution but they most often occur as the result of rubbing the contaminated hands and feet of the baby over other parts of the body, or from the clothing or possibly the attendant's hands. Every effort should be made to prevent the child's limbs from coming in contact with active lesions and the nurse should exercise great care not to touch unaffected parts of the body after dressing the lesions. The parts at the site of the lesions should be kept as dry as possible. Many mild cases will be best cared for by simple opening of the lesions and dusting heavily with stearate of zinc powder. Opening the lesions as fast as they occur and touching with 20 per cent. silver nitrate solution followed by applications of ammoniated mercury of from 2 to 5 per cent. works very efficiently in some cases. Iodin instead of silver nitrate has been used extensively but caution must be exerted to avoid iodin burns on the sensative skin of the newly born. Autogenous vaccins have been tried with seemingly little success. As a prophylactic measure vaccin may be found to have some value. As there is rarely any constitutional disturbance the usual hygienic and feeding schedules may be adhered to.

In the malignant cases treatment is of little or no avail. The blebs form and grow so rapidly that large areas of the body surface are soon denuded. Great difficulty is encountered in handling the baby because of these extensive lesions. It becomes impossible to use ordinary clothing, cotton wrappings being used to protect the child. Two per cent. ammoniated mercury ointment may be used freely or 20 per cent. silver nitrate solution if only applied to the periphery of the lesion. The vaccins have proved valueless in this type. Because of the constitutional disturbance and high temperature it is important to maintain the nutrition and body fluids. Danger to the mother and the difficulty in handling the baby makes it advisable in many cases to express the breast milk and feed the baby by tube, water being given in the same manner.

Ichthyosis Congenita

(Harlequin Fetus; Hyperkeratosis Diffusa Congenita; Cutis Sebacea; Seborrhea Squamosa Neonatorum)

In this rare condition the child is born with a marked ichthyosis, usually severe in character. The condition was first described in 1792. By 1895 Bowen had collected 42 cases, and some 54 authentic cases were reported in the literature in 1901. Most authors, prominent among whom are Ballantyne, Wassmuth, and Carbone, describe the disease as probably beginning about the fourth month of intra-uterine life and all are agreed that it is at least a perpetuation of a fetal condition into post-natal life. The fetal skin has a tendency to seborrhea, and ichthyosis congenita is an extreme example of this tendency to the formation of sebum or vernix. These children are very often prematurely born but the disease is not usually fatal during fetal life as very rarely has a case been reported in the stillborn. Hebra and Kaposi have considered all of these cases as a generalized seborrhea, but this view is questioned by Bowen and others, who contend that many of the mild cases are examples of the persistence of the epitrichial layer, which is usually cast off by the seventh fetal month but in these instances maintained its integrity up to the time of birth when it enveloped the infant like a distinct membrane. After a short time this membrane starts to peel off in large masses and sheets. These cases are not of course true ichthyosis but represent some type of the delayed physiologic shedding just referred to. Others are persistent vernix caseosa and possibly some are cases of dermatitis exfoliativa.

Riechke divides these cases into three forms. (1) True ichthyosis congenita. These are very severe, having intensive development in utero, the child dying in from one to four days. (2) Ichthyosis congenita larvota. These are milder cases which involve only part of the skin but are developed entirely or nearly so at birth. These children may live for a longer period. (3) Ichthyosis congenita tarda. In these cases there is no evidence of the disease at birth but it develops a little later. After all, the etiology of the condition is very obscure.

Pathology.—A considerable thickening of the corneal layer takes place, sometimes up to 500 microns as against the 34 to 47 microns of the normal skin, and this is augmented by a new formation in the rete and a rapid cornification producing profuse keratoma. Wassmuth found the changes almost entirely in the epidermis, the papillæ were much more numerous as well as broader and flatter, with great irregularity in form and size. The layers of the rete were thickened and the epithelial cells of the pegs assumed a spindle form, while nearer the surface they become polygonal. The granular layer could not be made out definitely except on the scalp. Sweat glands were greatly increased in number. Deformities of the sebaceous glands were caused sometimes by keratinization of the follicle-mouths. The hair was quite normal except for deformed shape. The cutis is usually normal but in some cases has shown a mild grade of chronic inflammation.

Symptoms.—The patient is usually weak and under-nourished, and often premature. The skin consists of large horny masses broken up by fissures and furrows. The entire surface of the body is covered with these fatty, thick, epidermal plates which are firmly adherent to the skin, the deep fissures extending down into the corium. The infant is enclosed as if in case armor and the face has a mask-like appearance. The mouth and its angles are distorted, causing a puckering and fissuring. Usually there is more or less ectropion. Deformity of the nose and ears are often present and the child being unable to suckle usually dies from inanition. In milder cases the membranous coating is more parchment-like giving the appearance of collodion or oiled paper and in these cases it is sometimes possible to keep the infant alive for a short period.

Prognosis.—In true cases the prognosis is usually very bad, the child living from a few days to a few weeks only. Recovery has been reported in a few very light cases.

Treatment.—Every attempt should be made to maintain nutrition and to keep up the body temperature. The application of oil or mild ointments in an effort to soften the skin is recommended but no curative measures have as yet been found.

NEVI

Nevi are usually described as being an affection of the skin, of congenital origin or disposition. They may be divided into two general classes. The first are those known as true tissue nevi which have originated from a proliferation of one particular portion of the tissue. Most of such nevi are elevated and more or less deeply pigmented. Some of the most common are nevi pigmentosis, simplex, verrucosus, spilus, and piliferus. The second class are those spoken of as organic nevi, which originate from a proliferation of the blood-vessels. The most commonly known of these is nevus vasculosus for which there are many names arising out of the type, color, shape, or location, as angioma, nevus sanguinius, nevus maternus, mother's mark, birth mark, nevus flammeus, port-wine mark, mulberry nevus, and others. A rarely described nevus, supposed to be due to a lack of capillary elasticity, is that known as nevus anemicus.

Etiology.—Nothing is known of the causes that lead to the development of the true or pigmented nevi, although heredity has been demonstrated in numerous cases. As to the development of the organic nevi a favorite theory advanced is the influence of maternal impression during pregnancy, which however has no support except in the minds of certain of the laity. The theory of intra-uterine pressure has been strongly advanced by Unna. He states that "the almost entire limitation of the congenital angiomata to the superficial layers would seem to point out that they are developed by the action of some external cause" and that the areas most often affected are those which are most likely to suffer from pressure during intra-uterine life. Virchow suggests a connection of the embryonic fissures of the skin with the appearance of the nevi, because of the abundant vascular development at the borders of the fissures, which he believes could be caused to proliferate rapidly from some slight irritative condition.

Pathology.—In the tissue nevi, microscopically, we find a proliferation of what is known as nevus cells in the cutis, a more or less marked deposit of pigment in the rete and the corium and very often a strong proliferation of the cones and papillæ. In the case of the organic nevi we may find a dilatation, or a new growth and hypertrophy of the vascular tissue usually limited to the corium but occasionally invading the subcutaneous tissues. In nevus anemicus Vorner found the blood-vessels smaller than in normal skin, the smaller arteries having the construction of capillaries, and the sweat glands atrophied or entirely absent toward the center of the nevus.

Symptomatology.—The pigmented nevi are rarely found to have so developed that they attract attention during the newborn period, the deposit of pigment and proliferation of tissue being so slight that it is usually overlooked during the early weeks of life. The organic nevi, however, of certain types especially those of the frontal and occipital regions are very much more prominent during the newborn period than later. Pollitzer found this type present in 35 per cent. of all newborn babes, while Depaul's observations at the Paris Clinic placed the number at one-third, and to those who have made careful routine examinations of any large number of the newborn, this percentage of occurrence would not seem too high. A further analysis by Geisler disclosed that 76 per cent. of the nevi of the newly born were in the region of the head, 3 per cent. on the neck, 11 per cent. on the trunk, and 9 per cent. on the extremities. Of those of the head, the occipital is by far the most common, with those of the frontal, the upper eyelids, the cheeks, ears, nose and lips following in frequency in the order named. The color of the nevi in the newborn is usually light red and those about the head are often easily overlooked except when the infant is crying. Most of the nevi of the frontal and occipital regions disappear the third month.

Prognosis.—Seldom is there any rapid increase in the growth of the nevi or the angiomata during the newborn period but, on the other hand, there is a decided tendency for the ordinary types to disappear.

Treatment.—It is rarely necessary to institute treatment of this condition during the first few weeks of life.

DERMATITIS EXFOLIATIVA

(Ritter's Disease; Dermatitis Exfoliativa Infantum; Keratolysis Neonatorum)

Dermatitis exfoliativa is a rare disease of the newborn occurring in most cases between the second and the fifth week, but occasionally seen as early as the first week. It is characterized by an intense wide-spread erythema which sometimes shows a tendency to a vesicobullous formation and is always followed by a marked exfoliation of the epidermis. Ritter collected in all 297 cases, first describing the disease in 1878.

Etiology.—In 1864 and 1865 when Ritter observed the first sporadic cases, they were thought to be pemphigus with an unusually severe exfoliation, but in 1870, during an epidemic in Prag, Ritter became convinced that the disease was a separate entity. Although the disorder is rare, much discussion has been aroused among interested authors since Ritter's observations especially as to whether dermatitis exfoliativa is not identical with pemphigus neonatorum, and many authorities believe that it is merely an excessive desquamation of a true pemphigus. Sperk, Winternitz, and Hazen have all found staphylococcus pyogenes aureus in the skin lesions and in one case in the blood of infants affected with the disease. This germ being the one most often found in pemphigus would seem to bear out the foregoing theory. Ritter's belief was that it was a dermatitis of pyemic origin. Kaposi believed that it was an exaggeration of the physiologic epidermic desquamation often seen in the newborn. Bohn classified the disease as a dermatitis without constitutional basis, while Luithlen, pointing particularly to the primary erythema, was of the opinion that it was produced by toxic factors. Escherich believed it to be a general septic infection. The general concensus of opinion, at present, however, seems to be that the exfoliation is secondary to a generalized staphylococcus infection of the skin which only becomes a general septic condition when the infection invades the blood stream of the infant.

Pathology.—A marked hyperkeratosis and an acanthosis within and without the cells is shown with the epidermis often raised from the papillary body by a bleb. There is a dilatation and engorgement of the vessels, edema of the papillary and subpapillary tissue with considerable small-cell infiltration. Hemorrhages are occasionally seen in the corium. Distortion occurs but no abnormalities are present in the organs of the skin. The stratum granulosum is often absent the pegs being chiefly involved.

Symptomatology.—The onset is sudden, usually between the eighth and twenty-sixth day, ordinarily beginning with a hyperemia of the skin of the lower half of the face, particularly the region of the mouth, becoming more and more intense, and spreading rapidly over the entire body. There is marked swelling around the mouth, particularly of the lower lip, the skin becoming thick and edematous, forming deep ridges and fissures which in severe cases make it impossible for the infant to open and close the mouth. Large blebs are apt to form over the body, lifting the corneal layer in large areas and exposing a moist, reddened rete, or in other cases, the entire trunk becomes red, dry and parched and the epidermis detaches itself and may be stripped off in large pieces. The process is apt to invade the buccal mucous membrane which becomes covered with greyish-white erosions. It may also in some cases even affect the cornea of the eyeball. The temperature as a rule remains normal and in some instances is subnormal. In mild cases the epidermis soon regenerates over the denuded areas, the desquamation ceases and recovery takes place, while in the more severe cases, the desquamation continues with a great loss of lymph and with probable secondary infection. The child rapidly becomes weaker and succumbs, as well from the great difficulty in maintaining nutrition with the lips and mouth so severely affected as from the process itself.

Diagnosis.—This condition is likely to be confused with pemphigus neonatorum and in certain border-line cases the picture may so closely resemble pemphigus that it is impossible to differentiate between the two conditions. This fact gives some grounds for the supposition that they may be the same infection, displaying somewhat different characteristics in different epidemics. The date of onset and the preliminary erythema, with the characteristic swelling about the mouth should serve to confirm the diagnosis in most cases. A bullous syphilid might simulate the skin picture of dermatitis exfoliativa very closely but the fact that this condition is apt to be epidemic, and that the affection would not present the other symptoms characteristic of hereditary syphilis such as snuffles, enlarged spleen, Wassermann reaction, etc., would serve to make the differentiation clear.

Prognosis.—The prognosis in this disease is grave. In Ritter's 297 cases about one-half terminated fatally. The difficulty in maintaining nutrition, the absence of normal skin function, and the probable development of a general sepsis are the factors which contribute to the gravity of the disease.

Treatment.—As in the case of pemphigus neonatorum the most painstaking and rigid isolation of the patient and cleansing of the ward should be carried out. Every effort should be made to prevent the loss of body heat in the patient, to maintain the nutrition, and preserve the strength by the least possible amount of handling. When applying treatment to the skin, there should be as little exposure as possible and the child should be immediately wrapped in cotton wool. Breast milk may be given with the Breck feeder or by gavage. The skin is kept clean by the use of sterile olive oil, applications of mild ointments or, in the less severe cases, a thorough dusting with stearate of zinc powder or equal parts of boric acid and starch. For the extensive lesions, dressings of 1 to 10,000 bichlorid solution, 1 per cent. ichthyol solution, or lime water liniments, are recommended.

INTERTRIGO

Intertrigo is a form of erythema occurring in those parts where the skin surfaces are in apposition. It is found most commonly in the newborn in the groins, the gluteal folds, the axillæ and the neck.

Etiology.—While a mild degree of intertrigo is common in the newborn, it seldom reaches the more aggravated form so often seen in older children. The hyperemic condition of the cutaneous surface is brought about by the decomposition of the skin secretions which are retained at body heat for considerable periods in the folds of the skin. In the gluteal region the condition is most often the result of irritation from feces or urine left too long in contact with the skin, or from the unwise use of irritating soaps. The skin of some children is much more sensitive than that of others and intertrigo is much more likely to occur when the skin is sensitive, and may act as a forewarning of eczematous tendencies. It is especially apt to appear when a child is over-nourished and the folds of the skin are deep.

Symptoms.—The skin of the parts affected is reddened and hyperemic with occasionally an actual inflammatory reaction. It seldom becomes macerated and there is no elevation or infiltration but occasionally the superficial layer of the skin will slough off. There is no elevation of temperature, and in the newborn the condition apparently causes little or no pain or discomfort.

Diagnosis.—There is little possibility of confusing this condition with any other in the newborn. The fact that it is confined to areas where the skin surfaces are in constant apposition, that it is purely local, and very transitory in character, make it easy to recognize. The fact that there are no blebs or pustules will distinguish it from pemphigus and allied conditions, which sometimes have their beginnings in these parts. The skin eruption of hereditary syphilis might simulate this condition, but would show a more severe degree of inflammation as well as other evidences of cutaneous syphilis and would not respond to treatment as readily as does intertrigo.

Prognosis.—In the newborn this condition responds readily to treatment, but is apt to recur if the exciting causes continue to exist.

Treatment.—The treatment consists largely of frequent and thorough cleansing of the affected parts, thus removing the cause of the irritation. Plain warm water or olive oil are probably best for this purpose. A very mild soap may be used if followed by clear water or oil to thoroughly remove it. The best results are obtained by keeping the affected parts thoroughly dried by the free use of a dusting powder, stearate of zinc being the one of choice. In the more severe cases Fuller's earth may be used successfully.

SEBORRHEA

The condition of seborrhea, more or less common in the newborn, is, at this age, undoubtedly a result of a functional disturbance of the sebaceous glands. At birth the skin is to a more or less degree covered by vernix caseosa, a cheesy deposit secreted by the sebaceous glands of the fetus mixed with exfoliated epithelium. The hyperactivity of the sebaceous glands in some children, resulting in a pouring out of an increased quantity of more or less chemically altered oily secretion, sometimes causes an irritation, with the result that seborrheic masses accumulate on the skin particularly in regions of the scalp and eyebrows, where the sebaceous glands exist in greatest number. Excessive washing or rubbing to remove this scale condition is likely to leave a dry, thin epidermis deprived of the normal amount of sebaceous secretion necessary for its protection and a dermatitis may result. It is very common in the newborn to see on and about the nose great numbers of dilated sebaceous gland ducts which appear to be plugged with solid white or yellowish fatty matter. These may also appear on the cheeks, forehead, lips and ears and have very occasionally been reported as spread over the entire body. There is no inflammatory reaction and the condition seems to be of no importance except that it emphasizes the fact of a functional instability of the glands.

Etiology.—Not much is to be said of the etiology of seborrheic conditions in the newborn. The bacterial theory of Sabouraud, however well it may apply to similar conditions in adults, certainly does not apply to the development of this condition prior to and so often present at birth. It is generally believed that these seborrheic conditions in the first weeks are due to functional instability which is aggravated by sudden changes in skin temperature, in pressure, and various other sources of external irritation, such as excessive bathing or rubbing, irritating soaps and the like. That there is an element of heredity must be admitted, as this tendency, probably exudative, has been frequently noted to be present in each newborn child in some families. Another factor which must be considered and one which probably has a direct bearing on the functional activity of the glands is that the condition is often aggravated by overfeeding although this is not as obvious during the newborn period as it becomes a little later.

Pathology.—According to Stelwagon the oily secretion found in the gland ducts and also on the surface, is chiefly composed of fatty matter. There is a dilatation of the sebaceous gland duct openings and this secretion is condensed to a fatty consistency. Microscopic examination shows many epithelial cells undergoing fatty degeneration and breaking down into granular débris.

Diagnosis.—Seborrhea in the newborn is not apt to be confused with any other condition. The oily nature of the scaly crusts, the location, and lack of inflammation are distinctive.

Treatment.—This condition is usually very mild during these early weeks and only the most non-irritating and soothing medication should be used. All that is usually found necessary is a thorough soaking of the parts affected with olive oil, mineral oil, or vaseline followed every two or three days by gentle washing with water and a non-irritating soap.

CHAPTER XX

PREMATURE INFANTS

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All infants born before the end of a normal term are born before the end of a full intra-uterine pregnancy, and consequently their organs are not fully developed, and as a result they must show certain definite body weaknesses, and a lack of resistance to the traumas of extra-uterine life.

This, however, is only a *relative body weakness* in the absence of inherited constitutional debility and malformations.

It is also a fact that the younger the fetus when leaving the uterus, the greater are the difficulties to be overcome in carrying out the required body functions necessary to life and therefore, the lower its vitality.

In a study of premature and congenitally debilitated infants at least two factors in the life history of the fetus must be considered.

1. The term of its intra-uterine life.
2. The state of its functional development at birth as evidenced by the presence or absence of inherited disease.

Classification.—For practical clinical purposes the group of infants comprising the prematures may be classified as follows:

1. Premature infants, with no pathological changes.
2. Premature infants, with pathological changes, due to:
   (*a*) Constitutional disease and chronic infections in the parents.
   (*b*) Maternal factors influencing the fetal nutrition, such as, overwork, under-nourishment, acute illnesses during pregnancy.
   (*c*) Local conditions in the mother.
   (*d*) Multiple pregnancies.
   (*e*) Constitutional defects and congenital malformations in the fetus.
   (*f*) Infants born to parents late in life.

Etiology.—The occurrence of premature birth depends upon many causes, which may be divided into those resulting in the expulsion of a healthy premature, and those which have a damaging effect upon the product of conception. In the first class may be included various injuries, falls, heavy lifting, overwork or other physical exhaustion; sudden emotional disturbances; premature rupture of the membranes, either accidental or intentional, when done in those conditions whose existence does not affect the nutrition of the ovum, as in pelvic and spinal deformity in the mother, placenta previa, etc.

Conditions in the mother requiring operative procedure not involving the uterine cavity frequently result in premature labor either through shock and trauma, resulting from operations as for ovarian conditions and uterine fibroids, or infections may be an added danger as in cholecystitis, cholelithiasis, appendicitis, ileus and renal operations.

The causes which fall within the second category all react to a greater or lesser degree upon the fetus, some producing only momentary weakness, as the milder acute infections, others causing a weakened physical condition as a result of their long continued action upon the nutrition and development of the fetus.

The most frequent causes are the chronic infections, of which *syphilis* plays the leading rôle. This condition in the mother is estimated as being a factor in a large percentage of all cases of prematurity, while Lesage and Kouriansky* state that syphilis is a factor in the causation of congenital debility of the full-term in 25 to 35 per cent. In a recent luetic infection, abortion is the rule; but as the disease becomes chronic, the succeeding pregnancies terminate later and later until a living child with or without manifestations of the disease is born, usually prematurely.

Chronic *nephritis* is one of the most frequent causes of spontaneous premature labor, and the offspring of the mothers are often puny either because of the systemic effect on the mother or resulting from impaired nutrition of the fetus due to placental hemorrhages and infarcts. Nephritis in the mother is also one of the most frequent indications for the induction of premature labor.

Pulmonary *tuberculosis* is less frequently the cause of premature labor, but the infants, even at full-term, are often undersized and weak. Congenital tuberculosis occurs but is rare. In the majority of cases, not the disease *per se*, but the predisposition is inherited.

Premature birth occurs in 30 or 35 per cent. of the cases of broken compensation in *heart disease*. The premature infants are, in these cases, often imperfectly nourished as a result of the poor aeration of the mother's blood. *Exophthalmic goitre* is occasionally the cause of premature emptying of the uterus. If chronic dyspnea exists as a result of laryngeal or tracheal stenosis from pressure, the development of the fetus will necessarily suffer.

Any of the *acute infectious diseases* may be responsible for the termination of pregnancy before the end of term. Pneumonia, influenza, typhoid fever, malaria, diphtheria, scarlet fever, measles, small-pox, Asiatic cholera, and bubonic plague, all have an influence on the duration of pregnancy. Prematurity is very common in pneumonia and influenza, especially when these diseases occur in late pregnancy.

Of *local conditions*, diseases of the decidua or endometrium, gonorrheal infection, and malpositions of the uterus frequently bring on premature labor, but usually before the fetus is viable. *Anomalous*

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* Lesage and Kouriansky, "Congenital Debility and Atrophy," Nourrisson, Paris, July, 1919, vii, No. 4, p. 193.

positions of the fetus in the uterus may be responsible for the premature expulsion of the uterine contents.

The occurrence of *multiple pregnancy* is a fruitful source of premature labor. Even when mature, twins are usually small and of low body-weight. This, of course, is even more true of triple pregnancies. In the presence of several fetuses the growth may proceed unequally so that one may be born with good vitality, and the others with greatly diminished strength.

Faulty nutrition of the fetus such as is found in overwork or from lack of sufficient food, as well as that due to wasting diseases, the blood dyscrasias (pernicious anemia, leukemia), and *intoxication* from alcohol (acute and chronic), phosphorus, arsenic, mercury, or lead, may cause either an early termination of pregnancy or so serious a lowering of nutrition of the fetus that the vitality at birth may be greatly impaired. In addition, congenital malformations in the fetus sometimes bring on premature birth. In *diabetes* prematurity is not infrequent, and the infants may show glycosuria.

Infants born to parents late in life are predisposed to prematurity, perhaps because of the factor of undernourishment. This is also the case in prematures born of women who have had numerous pregnancies at short intervals.

Finally, habitual miscarriage resulting in the interruption of successive pregnancies, not infrequently at about the same stage, without evident cause is not rare. The author has records of several such women in whom there is no history of syphilis or other constitutional disease, and in whom uterine deformity is not demonstrable, the cause remaining unknown.

PHYSIOLOGY

Clinical Features.—The appearance and characteristics of the *healthy premature* child vary with the fetal age at the time of birth. With a lengthening of the period of gestation, the distinctive characteristics of the fetus become less and less marked until it becomes impossible to differentiate the slightly premature from the full-term infant. Premature infants may exhibit the following features in varying degrees.

The body is usually small and puny, though in some instances the infant may be of a considerable size, yet with a very imperfect development of its internal organs.

The weight is low, varying from amounts approximating 700 gm. (1½ lb.) to 2500 gm. (5½ lb.), in the viable. The latter figure may be exceeded by infants nearing maturity, and by some of the full-term weaklings, but will serve as a fair maximum.

The skin is soft and varies from pallid to a vivid red. The epidermis is thin and the blood-vessels are easily visible. The skin frequently hangs in folds. The adipose tissue is scant, the features are angular, and the infant looks old. Lanugo is plentiful, especially upon the extensor surfaces of the extremities.

The skull is round or ovoid in contra-distinction to the usual markedly dolichocephalic skull of the full-term newborn. The fontanelles are large and the sutures prominent.

The nose exhibits many small comedones. The ears are soft and small and hug the skull.

The nails have scarcely reached the ends of the fingers even in the larger infants, while in the smaller infants they may be very poorly developed.

The cry is feeble, monotonous and whining. The infant lies in a deep sleep, and must be aroused for its feedings. Efforts at suction are weak or absent. All movements are slow, all functions are sluggish, and the child shows a pronounced degree of muscular inertia. The temperature has a very decided tendency to remain below normal and is inclined to be irregular in character. The urine is usually scanty. The bowels are sluggish and constipation is the rule. Early and intense jaundice is common. In this early stage they may be described as the "Oldman Type."

These are the principal findings which are to be seen on superficial examination. It must be remembered that any of these symptoms may vary in different individuals of the same age, depending upon the cause of prematurity, and upon the condition of health present in both the mother and the child.

Infants of good vitality, with proper hygienic surroundings and nursing care and a sufficiency of good breast milk, soon assume the "*Doll Type*" appearance. The head enlarges (megacephalus, to be described later), the face fills out due to the deposit of thick fat layers and the buccal cushions become especially prominent, while all wrinkles disappear. Large veins cross the forehead and temple regions. The skin becomes smoother, and although usually pale the cheeks may show pink, and the small nose and white sclera add to the attractiveness of the infant's appearance.

Characteristic of the second quarter-year of life in many babies is the relatively large tongue which is often seen protruding from between the open lips. This feature often gives the face an expression of the adenoid type. The small nares, which are easily obstructed by even a slight congestion and especially by posterior rhinitis, are an added factor in the development of this expression.

At about this same period many infants develop an exophthalmos, probably because the skull cavity is not large enough to accommodate the enlarged brain. The sclerotic coat of the eyes may be seen beyond the cornea and the pupils are large.

The determination of the exact *age of the infant prematurely born* is a matter of considerable difficulty. The information furnished by the mother as to the time of her last menstrual period, or as to the time when life was first felt, gives an entirely unreliable approximation of the probable date of confinement, and errors of a month or even more are not rare. In institutions for foundlings all data are as a rule absent, and other methods for determining the infant's fetal age must be relied upon exclusively. The weight of the infant is of uncertain value also, as an infant of 1500 gm. weight, may be the product of a pregnancy of seven months in a healthy woman, while one of the same or less weight may be the eighth-month offspring of an albuminuric or syphilitic mother. The body measurements also vary materially with the individual. The degree of development of the osseous system is of great value in determining the anatomical development, and indirectly the condition of the bones acts as a guide to physiological development even though they do not give absolute data as to age.

More important than a determination of the approximate term of pregnancy or a consideration of the size of the infant, at least in those infants born but a few weeks before the natural termination of the period, is a history of syphilis, tuberculosis, traumata, or other causes, operating in the mother and responsible for the early emptying of the uterus.

Body-weight and Other Measurements.—Infants born at full-term weigh on the average from 3000 to 3500 gm. The dividing line between the premature and full-term infant has been generally placed at 2500 gm. If under that figure, they may be considered below normal as far as concerns the strength and ability to overcome the forces which assail them on every hand. The weight of the premature varies even within greater limits than that of the full-term infant, and as one may see a child below 2500 gm., so also there are prematures with a weight above this limit.

The weight depends upon the cause of the premature birth, and upon the age of the child. Those born of mothers afflicted with nephritis, tuberculosis, or other wasting diseases, and infants showing active syphilis, are usually considerably smaller than the same aged infants of healthy parents. Diseases and abnormal location of the placenta also restrict the growth and retard the weight of the fetus. The infant in placenta previa is often undersized, even when born at term. Multiparity may predispose to undersize.

The weight and length as compared to the fetal age is shown in the following table from Oberwarth:*

Fetal age, weeks	Weight, grams	Length, centimeters
26	330 to 1041	28.0 to 37.0
28	995 to 1408	36.3 to 37.5
30	797 to 1700	33.1 to 41.3
32	1868 to 1964	42.0 to 42.7
34	1286 to 2213	39.0 to 47.0
36	2424 to 2700	46.1 to 48.0

* Oberwarth, Ergeb. d. inn. Med. u. Kinderh., vii, 191.

These compare favorably with those give by Anhlfeld and Hecker.*

Fetal age, weeks	Weight, grams	Length, centimeters
27	1140	36.3
29	1575	39.6
31	1975	42.7
33	2100	43.9
35	2750	47.3
37	2875	48.3

Potel and Hahn's† figures do not include the length.

Fetal age, weeks	Weight, grams
27	995 to 1146
29	1540 to 1700
31	1881 to 1964
33	2150 to 2213
35	2400 to 2700

A group of the author's cases which follow give the age of the fetus as computed from the date of the last menstruation. That this is an unreliable method may be recognized by noting the variation in figures in cases 2, 3, 11, 14, 15. In the other instances the figures compare well with those of the above.

	Fetal age, weeks	Weight, grams	Length, centimeters	Diameters of head				
				O. F.	Bi. P.	Bi. T.	Oc. M.	S. O. B.
1	21	700	30.0	7.5	5.5	4.5	9.0	7.5
2	22	1015	37.0	7.5	6.5	6.0	9.0	7.5
3	27	1690	40.0	9.0	8.0	6.5	11.0	7.5
4	29	1449	....	8.0	7.0	7.0	8.0	7.0
5	31	1175	37.5	9.0	7.0	6.0	11.0	8.0
6	32	1380	34.0	9.0	8.0	7.0	11.0	7.0
7	32	2040	45.0	11.5	8.5	7.5	13.0	9.5
8	33	1175	44.0	9.0	7.0	6.0	11.0	8.0
9	33	2110	45.0	10.0	8.0	6.0	12.0	8.0
10	38	3625	50.0	11.0	9.5	8.0	13.25	9.5
11	39	1610	41.5	10.0	7.75	6.25	11.75	8.5
12	39	3260	49.0	11.5	9.5	8.5	13.5	9.75
13	40	1370	38.0	9.0	7.0	6.0	10.0	8.0
14	41	1570	35.0	11.0	8.0	7.5	11.5	7.0
15	42	1810	38.5	10.0	8.0	7.5	12.5	8.5

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* Ahlfeld and Hecker, Arch. f. Gynak, ii, 1872.

† Potel and Hahn, De l'accroissement en poids des enfants nés avant terme, Thèse Paris, 1895.

In contrast with these measurements of the diameters of the head in prematures, the average measurements of the skull in a mature newborn are noted as follows by Schauta:*

1. Diameter suboccipito-bregmaticus (from the posterior edge of the great occipital foramen to the anterior angle of the great fontanelle) 9 cm.
2. Diameter fronto-occipitalis (from glabella to the occipital protuberance) 11 cm.
3. Diameter mento-occipitalis (from the point of the chin to the farthest point of the occiput) 13 cm.
4. Diameter verticalis (from the vertex to the base of the skull) 9.5 cm.
5. Diameter biparietalis (between the parietal tuberosities) 9 cm.
6. Diameter bitemporalis (between the farthest points of both coronary sutures) 8 cm.

The doubtful cases for classification are those which range from 2000 to 2500 gm. in weight and 45 to 48 cm. in length.

One cannot estimate the vitality of these infants from a consideration of their birth weight. Many of them born at or near term have a normal weight, yet they do not survive. On the other hand, infants of considerably less weight may present evidence of great vitality, a lusty cry, and take nourishment with avidity. According to our experience the condition of the turgor of the prematurely born infants is of much more importance than all these. Flabby prematures with poor turgor and decreased tonus are usually not viable. Prematures with good turgor and tonus even with a low weight more frequently survive.

In addition to the variations in weight and length, the premature shows variations in other measurements, and a comparison of several measurements in the same individual are of more value than a consideration of one measurement alone. The circumference of the head is always greater than that of the shoulders in the normal premature. They also show relatively greater length of the upper half of the body as compared with the lower extremities, the latter growing especially fast during the second half of the intra-uterine existence.

Von Winckel† regards the circumference of the head as of importance for the diagnosis of the age of the fetus and gives the following figures:

4th month	10–14 cm.
5th month	13–18 cm.
6th month	19–24 cm.
7th month	23–28 cm.
8th month	25–30 cm.
9th month	29–33 cm.
10th month	32–37 cm.

Reiche‡ reports the following comparative body measurements:

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* Schauta, F., Lehrb. d. ges. Gyn. ii, Aufl. Leipzig u. Wien., 1897.

† Von Winckel, Lehrb. d. Geb., Leipzig, 1889.

‡ Reiche, A., The Growth of the Prematurely Born in the First Months of Life. Zeitsch. f. Kinderh. Orig. xiii, 332, December, 1915.

TABLE 1

Group 1	12 children		Weight 800 to 1200 gm., average
	Minimum	Maximum	
Length of the body	34.0 cm.	41.0 cm.	37.4 cm.
Circumference of chest	21.0 cm.	24.5 cm.	22.5 cm.
Circumference of head	24.0 cm.	29.5 cm.	26.8 cm.
Group 2	**26 children**		**Weight 1200 to 1500 gm.**
Length of the body	37.0 cm.	45.0 cm.	41.6 cm.
Circumference of chest	22.5 cm.	27.5 cm.	24.8 cm.
Circumference of head	26.0 cm.	31.0 cm.	28.4 cm.
Group 3	**28 children**		**Weight 1500 to 2000 gm.**
Length of the body	41.0 cm.	48.5 cm.	44.2 cm.
Circumference of chest	25.0 cm.	32.5 cm.	27.2 cm.
Circumference of head	27.0 cm.	32.0 cm.	30.3 cm.
Group 4	**22 children**		**Weight 2000 to 2500 gm.**
Length of the body	41.5 cm.	49.0 cm.	46.5 cm.
Circumference of chest	26.0 cm.	30.0 cm.	28.4 cm.
Circumference of head	29.0 cm.	33.5 cm.	32.2 cm.

These figures show a gradual and steady increase of the weight, the chest and head measurements, up to the time of maturity, when they should average 3200 gm. in weight, 50.5 cm. in length, with a chest circumference of 32.9 to 33.8 cm. and a head circumference of 34.5 cm.

We see in the eighth to tenth month an abrupt rise of the curve of chest circumference, the curve flattening somewhat soon after the birth. This increase in the circumference of the chest in the last fetal months is considerably greater than that of a mature child during the first months after birth. In the latter the circumference of the chest increases from 32.5 to 37.2 at the end of the third month to 41.0 at the end of the sixth month, therefore, in the first six months of life, approximately about as much as in the last three fetal months.

In the curve of the growth of the skull the flattening appears even somewhat earlier. The ratio, however, between the growth of the skull in the last three fetal months and that in the first six months of life, is the same as in the circumference of the chest. Also the circumference of the head grows absolutely and relatively considerably more in the last fetal months than in the first six months of life, since in this time it grows only from 34.5 to 42.7 cm.

Ylppö* recently studied the relation of the chest circumference to that of the head in prematures and full-term infants. He found that at birth the circumference of the head is greater than that of the chest, and the greater the prematurity the more marked is the relative disproportion between the head and the chest circumferences. These facts are borne out by his table:

Weight of infants, grams	Number	Circumference of head, centimeters	Circumference of chest, centimeters	Breast circumference, per cent. of head circumference
Under 1000	16	25.0	20.8	83.2
1000–1500	78	31.8	24.5	77.0
1501–2000	75	30.0	26.3	87.7
2001–2500	74	32.3	29.5	91.3
Newborn 3000–3500	100	33.5	31.0	92.5

Variations in Osseous Development.—There are, as might be expected, some variations in the normal process of ossification, which may be the result of pathologic conditions of the mother and of the fetus (syphilis, rickets, osteogenesis imperfecta, etc.). In general, these pathologic processes may be diagnosed in the roentgenograms. In some portions of the skeleton the ossification is less regular than in others, and as a general rule the parts of the skeleton below the dorsal region are more subject to variations in the process of ossification; and the centers which develop at a later period of fetal life are also more variable. (See Fig. 84 p. 260, Vol. I.)

Values of the Different Portions of the Body.—In the very early period (second month) the stage of ossification of clavicle and mandible is of chief importance, and on the basis of presence or absence of these centers determination of the age is made.

Next in importance are the centers of the upper extremity, and especially of the hand (metacarpals and phalanges) which are very regular, not only in the time of their appearance, but also in their sequence. The ossification of the diaphysis of the long bones of the arms extends from the eighth to the sixteenth week, and during this period the determination of the age may frequently be made from a good roentgenogram of the hand alone.

The progress of ossification of the head is also of considerable diagnostic importance, but the centers in many bones of the head are very difficult to demonstrate. Those, however, that can be well demonstrated are of much value in the determination of the age. This is especially true of the occipital bone, superior maxilla and tympanic ring.

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* Ylppö, Arvo., Pathologisch-Anatomische Studien bei Frühgeburten, Zeitschr. f. Kinderh. Orig. Bd. xx, Mar. 25, 1919.

The axial skeleton (the vertebral column) is less reliable than the foregoing named portions of the skeleton, and especially its lower portion is of little value in diagnosis of age. It is not the absolute number of arches or of the bodies ossified which decides the diagnosis of the age of the fetus, but more the region involved and the extent of the development in the particular region of the vertebral column (cervical, dorsal, lumbar, sacral). On the other hand, however, the facts that the process of ossification of the vertebral column extends from the ninth week throughout the life of the fetus, and all its centers, as a rule, are well demonstrable, make it of especial value for at least approximate determination, although it must not be forgotten that occasionally the process of ossification may be delayed in the vertebral column, while it is normal and regular in other portions of the body.

The sternum is unreliable as an index of age and its centers are frequently difficult to demonstrate. The ribs are fairly constant, except that the twelfth pair may not show at all in roentgenograms of comparatively old fetuses.

While the ossification of the long bones of the legs are fairly regular, since they appear at an early period, ossification in the foot is very irregular and the stage of ossification of the foot is of little value in the determination of the age of the fetus. The osseous development of the foot extends from the ninth week to the end of the fetal period and into extra-uterine life. Throughout this time there are very marked variations, especially in the centers which appear late in the fetal period.

From what has been said it may be seen that, as a general rule, the earlier a center appears the more regular it is, and since the process of ossification starts in the cephalic region and spreads caudally, it is also true that the more caudad a skeletal segment is situated the more it is subject to variations and irregularities.

Cause and Nature of Hypothermia.—Heat regulation is one of the least developed functions of the premature infants, the body temperature showing marked fluctuation with a tendency to hypothermia. This is due to several factors:

1. *Faulty Heat Regulation Due to Lack of Development on the Part of the Nervous System.*—It is possible to imagine that in a premature infant in whom the development of the brain is still going on, and in whom the separation into the white and grey matter has not been completed that the nervous system is not sufficiently matured to functionate normally.

2. *Loss of Heat through Radiation.*—The extent of the heat loss from the body of an animal by conduction, radiation, evaporation from the skin is determined by the extent of the surface and by the thickness of the poorly conducting subcutaneous fatty layer; the heat

loss, therefore, is in greater part proportional to the extent of the surface of the body. Heat is also carried away in expired air from the surface of the lungs. In a premature infant the body surface is relatively greater than in a full-weight newborn, since the size of the body is absolutely smaller. Wrinkled skin and absence of fat deposits in the skin are responsible for the greater loss of heat. It is these physical conditions which make it difficult for the premature to retain its own heat and predispose to subnormal temperature.

3. *Insufficient oxygen combustion* due to the poorly developed respiratory center and the tendency toward asphyxia.

4. *The circulation* as affected by its undeveloped nervous mechanism and weak cardiac action is another important factor.

5. *Insufficient Heat Production Due to Lack of Food or Improper Metabolism.*—This cause of hypothermia is of minor importance in the premature infant which is fed a sufficient quantity of breast milk and shows ability to assimilate the same. As the sucking centers are too poorly developed to obtain a sufficient diet, most of these infants cannot be trusted to their own resources in obtaining their food.

A careful consideration of all of the factors tending to hypothermia make it evident that we cannot depend on an equalization of the heat loss from the body surface by the internal production of heat, and it becomes evident that in order to maintain a uniform temperature it becomes necessary to artificially assist the infant by giving it an environment of good air sufficiently heated to maintain a normal body temperature.

The Growth of the Premature.—*Initial Weight Losses.*—Loss of body-weight during the first days of life occurs so constantly in full-term infants that moderate losses must be considered physiological. This is also true of premature infants although in most instances it is relatively greater. Premature infants lose relatively more and regain their birth-weight slower, while the nearer the prematures are to full term, the lower is the relative loss of weight as expressed in percentages.

The average loss of weight in the premature and in other infants of relatively low birth-weight during the first days of life is shown in the following table adapted from Reiche.*

Weight, grams	Length, centimeters	Average decrease, grams
800–1200	32–40	71
1200–1500	37–44	97
1500–2000	40–48	137
2000–3500	41–50	177

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* Reiche, Ztschr. f. Kinderhk., xii, 1914, p. 369.

Gundobin's* figures are considerably higher, he having come to the conclusion that the initial loss of weight in infants with a birth weight under 2000 gm. amounted on the average to 148 gm.

Artificially fed infants lose more weight than breast fed, but no differences were noticeable between those infants nursing at the mother's breast and those fed by a wet-nurse (Reiche†).

In children of multiparous women both the absolute and also the relative percentage value of the weight loss is smaller than in those of primiparous, which is undoubtedly due to better nursing conditions, and to the fact that milk appears sooner in multiparæ and is usually more abundant.

The loss of weight is also relatively larger the less the birth weight of the infant, as the following table taken from Pies‡ will show:

Initial weight, grams	Primiparæ, average decrease	Multiparæ, average decrease
2500	240 grams = 11.2 per cent.	195 grams = 8.2 per cent.
2510–3000	235 grams = 8.3 per cent.	180 grams = 6.2 per cent.
3010–3500	295 grams = 9.0 per cent.	265 grams = 8.1 per cent.
3510–4000	360 grams = 9.7 per cent.	325 grams = 8.7 per cent.
4010–4500	245 grams = 8.4 per cent.	366 grams = 8.3 per cent.
Average............	275 grams = 9.3 per cent.	266 grams = 7.9 per cent.

Gundobin§ found that the lowest weight was usually reached sometime between the fourth and sixth days in the full-term infant and that the birth weight was regained on the eleventh to sixteenth day. Very frequently, however, and especially in weaklings and prematures, the birth weight is not regained as early as the sixteenth day, 20 or 30 days being required to make up the initial loss. The artificially fed regain the loss later than breast fed infants.

Pfaundler|| in his observations on 1000 newly born infants came to the conclusion that in 42 per cent. the physiological weight loss occurred by the fourth day. The loss in the infants of from 1500 to 4000 gm. birth weight averaged 7.8 per cent. of the latter, and was about the same for the heavy as for the light, although it was relatively slightly greater in the former.

* Gundobin, Besonderheiten des Kindersalters, Berlin, 1912.

† Reiche, Ztschr. f. Kinderhk., xii, 1914, p. 369.

‡ Pies, Uber die Dauer die Grosse und den Verlauf der physiologischen Abnahme des Neugeborenen, Monatschr. f. Kinderh. ix, 1911, p. 51.

§ Gundobin, *Ibid.*

|| Pfaundler, Körpermass-Studien an Kindern, Zeitsch. f. Kinderh., Mar. 28, 1916, pp. 151–2.

TABLE SHOWING WEIGHT CURVES OF A GROUP OF BREAST FED PREMATURE INFANTS

(Author's Cases)

Cases	Weight on admission	Age on admission, days	Initial loss	Percentage loss
1. Baby H.	840	1	200	24.0
2. Silvis	1050	3	70	7.0
3. Juniata	1070	1	80	8.0
4. Allan B.	1135	7	35	3.0
5. Grace A.	1180	3	110	10.0
6. Peter P.	1175	1	220	20.0
7. Peggy	1185	..	0	0.0
8. Joseph R.	1190	3	40	3.4
9. George	1335	1	60	4.5
10. Glenn	1340	..	155	11.5
11. Ann C.	1340	18	0	0.0
12. Ethna B.	1360	1	0	0.0
13. Edward R.	1360	3	80	6.0
14. Grace H.	1365	2	125	9.0
15. Grace B.	1440	1	30	2.0
16. Nell H.	1420	1	0	0.0
17. Hattie	1435	1	185	13.0
18. Marie B.	1750	1	130	7.0
19. Marie G.	1765	1	170	10.0
20. Edyth	1875	1	85	4.5
21. Martin Jo.	1890	1	40	2.1
22. Stella	1930	1	0	0.0
23. George	1935	1	300	15.0
24. Jim	1950	1	0	0.0
25. Josephine	1965	1	60	3.0
26. Mike	2010	1	0	0.0

Table Showing Weight Curves of a Group of Breast Fed Premature Infants

(Author's Cases)

Calories and Daily Gain

Admission weight regained, days	Weeks in hospital	Weight when discharged	Average calories fed after admission weight was regained	Average daily gain after admission weight was regained
37	27.5	1950	95–263	7.0
17	9.0	1580	91–142	11.0
15	15.0	2180	132–170	12.5
15	9.0	1655	96–112	10.6
36	11.5	1875	105–145	17.0
32	15.5	2480	107–150	17.0
No loss	6.5	2155	105–155	21.6
12	8.0	1950	86–144	17.0
6	4.0	1625	95–161	14.0
28	16.0	3245	112–160	24.0
No loss	17.0	3265	120–166	16.3
No loss	12.0	2512	137–220	13.0
14	8.0	2100	82–126	18.0
20	10.0	2510	95–160	22.0
6	15.0	2960	109–136	14.3
No loss	8.0	2940	115–144	27.0
26	6.0	1650	116–148	13.0
20	6.0	2225	102–119	21.5
7	6.0	2120	114–125	9.0
5	6.0	2615	104–120	23.0
3	5.0	2515	127–170	14.0
No loss	5.0	3160	105–131	37.0
33	10.0	2835	120–171	25.0
No loss	5.0	3315	111–120	40.0
10	5.0	2635	105–132	23.0
No loss	6.0	3775	94–118	43.0

Shick* believing that the initial loss of weight was avoidable, gave each infant 10 per cent. of its body-weight of breast milk the first 24 hours, increasing the amount until 15 per cent. was given at the end of the third 24 hours. He employed the milk of mothers having infants less than a week old and was able to prevent the initial loss in all of his 12 cases.

Cammerer† compared the increase in weight in breast fed and bottle fed premature infants with an initial weight of from 1590 to 1740 gm.:

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* Shick, B., Zur Frage der Physiologischer Korpergewichtsabnahme des Neugebornen, Ztschr. f. Kinderh. xiii, 1916, p. 257.

† Cammerer, Med. Wochscrft., 1909, no. 34.

	Doubled weight	Trebled weight	Quadrupled weight
Breast fed	10th week	22nd week	33rd week
Bottle fed	11th week	24th week	40th week

The average daily increase in weight of the premature of different periods as well as for the premature child is shown by Friedenthal:*

Fetal months	Average daily increase in weight, grams
6th to 7th	19.5
7th to 8th	29.3
8th to 9th	23.3
9th to 10th	13.3
1st month of mature child	25.0

The growth in length proceeds slowly from month to month, diminishing slightly in rate (Friedenthal).

Age	Growth in length per month, centimeters
6th to 7th fetal month	6.0
7th fetal month	5.0
8th fetal month	4.5
9th fetal month	4.0

If these figures of Friedenthal's are plotted into a curve it is seen that the curve of the body-weight and that of the body-length run parallel up to the seventh or eighth month, at which time the length curve rises less abruptly than the weight curve.

Pfaundler† found that the rate of growth in an infant born three months prematurely became the same as that of a maturely born child when the premature had reached the age of three months. These figures apply, of course, to the healthy prematures only and not to those debilitated from disease, improper food, or by unfavorable environment.

Reiche‡ has also studied the relation between the growth in weight and the growth in length and has introduced the term length-weight coefficient, by which is understood the weight of a unit of length.

* Friedenthal, Deut. Med. Wochscrft., 1909, No. 34.

† Pfaundler, Ztschr. f. Kinderhk., xiv, p. 1.

‡ Reiche, Ztschr. f. Kinderhk, xii, 1914, p. 369.

The following table shows the birth weight coefficient for different groups of prematurely born infants:

Birth weight, grams	Length of body, centimeters	Length-weight coefficient, grams
800–1200	32.0–40	28.0
1200–1500	37.0–44	33.8
1500–2000	40.0–48	43.2
2000–2500	41.5–50	48.7

Langstein* formulated the following law from the observations of Reiche and others: Both the growth in mass and the growth in length of those organisms in whom the transition from intra-uterine to extra-uterine life had to occur prematurely, proceeds according to the same laws that correspond to the period of time after impregnation.

Weight in Relation to the Body Surface.—Ssytcheff† gives the following table comparing the surface area and the weight in the premature and in older children.

Age	Weight, grams	Surface area, square centimeters	Surface area per kg. of weight, square centimeters
Premature 4 days old.	1505	1266.4	841.4
Newborn.............	2097	1476.0	704.0
15 days old...........	2980	2129.0	711.0
6 months old.........	5138	2961.0	576.2
1 year old............	9095	4800.0	527.0

Thus it is seen that the larger the volume (weight) of the infant the smaller the surface area relative to that weight.

PATHOLOGICAL FINDINGS IN PREMATURES

Premature infants must be classified into two groups: Those that are born "weaklings" due to congenital deformities or malformations, congenital diseases, especially lues; and the congenital weaklings born of nephritic, eclamptic or tuberculous mothers, or those suffering from chronic toxemia. In the second group are those fully developed and normal for their fetal age.

One is often amazed at the life energy of these prematures, in view of the high grade pathological changes in the various organs, especially the hemorrhages into the brain and spinal cord.

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* Langstein, Ernahrung und Wachtsum Frühgeborener, Berl. klin. Wochschr. 1915, p. 24.

† Ssytcheff, Zitiert nach Gundobin. Die Besonderheiten des Kindesatters, Berlin, 1912. Allg. mediz, Verlangsantalt.

Premature birth should be considered a traumatic process, in which the characteristic pathological processes are most frequently noted in three groups of organs, for which there appears to be a predilection:

1. The skull with the brain and its membranes, inclusive of the spinal cord.
2. The lungs.
3. The gastro-intestinal canal.

Intracranial hemorrhages are especially important. Ylppö* believes that they are responsible for 30 per cent. of the deaths of prematures in the early days of life. In the skull, there are subarachnoidal or intrapial hemorrhages, while in the spinal column they are extradural.

The so-termed subdural hemorrhages and those from tears of the tentorium, which are present in full-term infants, are only exceptionally seen in prematures.

Ventricular hemorrhages are frequently found in prematures, but hemorrhage into the brain substance proper is quite rare. High grade edema of the pia is as a rule also present along with these intracranial hemorrhages. Bacteria easily settle in the injured brain membranes and meningitic processes are not uncommon.

In the later life of the premature, the appearance of spastic states (Little) and of disturbances in intelligence is often seen and is explained as a rule, as a consequence of old hemorrhages into the brain and spinal cord.

The term megacephalus (Ylppö) is applied to the condition in which the development of the head is out of proportion to the other body measurements. The head is abnormally large and the brain is also relatively larger. This condition is a characteristic in a high percentage of premature infants and occurs in inverse proportion to the fetal age and birth weight.

Megacephalus usually occurs before the age at which rachitic changes are noted in the long bones and chest. However, as rickets occurs much earlier in premature infants than in those born at full term I do not believe that at present we can dissociate these two conditions. It is therefore probable that the same etiological factors causing the development of megacephalus in the first months may cause the rachitic manifestations in the bones and other organs at later periods.

Hemorrhages into the lungs appear not only under the pleura, but are scattered through the entire parenchyma. The alveolar septa are thickened because of the extravasations of blood. The normal circulation of the lung is hindered and in the extra-uterine life there appears a stasis, which hinders the taking up of air and predisposes secondarily to atelectasis. Following stasis and bacterial changes, there may appear in the lungs of prematures a high grade, almost total, inhibition of the circulation of the blood.

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*Ylppö, Arvo, Zeitschr. f. Kinderhlk., xx, 1919, p. 212.

Inflammatory changes in the lungs or bronchi appear infrequently and atypically in the first days of life. Bronchopneumonia, after the second week of life, begins to play an important part in causing death.

Epicardial hemorrhages have little to do with life. They appear just as the subpleural hemorrhages and those in all the other organs.

Subcapsular liver hemorrhages are comparable with other subserous hemorrhages. They are important only in so far as they may be extensive, and with rupture of the capsule may result in hemorrhage into the peritoneal cavity with death.

Hemorrhages into the kidney are frequent. They have two predilections: in the interstices of the apices of the pyramids; or in the neighborhood of the *venæ et arteriæ arciformes*. Hemorrhages into the Malpighian bodies and cortex are rare. Infarcts appear in the same sites as the hemorrhages.

The hemorrhages of the digestive tract are next in importance to those of the brain. In small prematures, dying shortly after birth, one finds hemorrhages scattered through the entire tract. The areas of predilection are: the lower portion of the esophagus; the cardia and the fundus of the stomach; the mucous membrane folds in the corpus ventriculi; the duodenal margin of the pylorus; and the entire duodenal mucosa. In the deeper portions of the intestines, hemorrhages occur infrequently about the ileocecal valve and in the mucosa of the large bowel. These hemorrhages appear chiefly under the epithelial cells in the tunica propria. Blood often appears in the bowel lumen after rupture of the mucosa. These hemorrhages in extra-uterine life are important only in so far as they predispose to infection, which occurs easily. As a result, within one and a half days prematures may show a marked mucous membrane necrosis and peritonitis.

Inflammatory processes within the digestive tract, especially in the esophagus and duodenal mucosa are also common. From the inflamed intestinal mucosa, bacteria easily invade the blood stream with subsequent enteral sepsis.

The mucosa of the stomach must also be given the greatest consideration in the frequent septic processes of the prematurely born, especially during the first few days of life. Involvement of the stomach is often followed by peritonitis and by bacillus coli septicemia.

The histopathologic inflammatory processes due to bacteria appear atypically in prematures. This is associated with a very ineffective exudation of fibrin and scanty mobilization of leukocytes. Because of these factors general sepsis in all infections appears easily.

The hemorrhages are due to diapedesis, rhexis, or both, also to the intensity and duration of stasis or to the grade of the infectious—toxic—damage to the capillary walls.

The preceding summary of the pathological changes in the premature has been concerned chiefly with the question of hemorrhage. Specific pathology will be discussed later under the "Diseases of the Prematurely Born."

CARE AND NURSING OF PREMATURE INFANTS

To be successful in the care and nursing of these little patients a certain routine must be followed:

1. Preparation for their home or hospital care must be made, whenever possible, before labor begins.
2. Their immediate care after birth is of greatest importance.
3. The general care must be adapted to their individual needs.
    (*A*) Refrigeration must be prevented.
    (*B*) Skilled nursing is essential.
4. Human milk must be provided for those born before the thirty-sixth week.
5. The daily routine must be adapted to the infant's age and development.
6. Contact between the infant and individuals not concerned in its immediate care must be avoided. Attendants ill with colds and other forms of infection should observe most rigid rules of asepsis to avoid cross infections.

Preparations for the Infant's Birth.—In case of expected premature abor immediate preparations must be made for the reception of the infant into a proper environment. The preparation must not be delayed until labor has begun, otherwise many viable premature infants will be lost. If the proper facilities cannot be furnished in the home, the mother should be persuaded to enter a hospital before confinement. She should be impressed with the fact that every day of added intra-uterine life will improve the infant's chance not only for life, but also for normal development.

Immediate Care of the Premature Infant. *Asepsis.*—The greater susceptibility of the prematures, especially of their less protected skin, demands even more painstaking observation of rules that hold good for newborn infants in general. These infants are much less resistant than are the full-term and succumb more readily to infection.

Preservation of Body Temperature.—The preservation of heat must be begun immediately after birth of the infant, preferably on the confinement bed itself, as the extent of the initial temperature drop is of no mean consequence to a premature infant. After severing the cord the infant should be placed in a heated basket or incubator bed, which should be a part of the equipment of the delivery room.

In the home, hot-water bottles, a properly protected electric pad, (page 487), or an improvised incubator (page 486) will answer the purpose. It should be remembered that these infants are easily burned and such burns are usually fatal.

In small prematures the cotton-pack, completely enveloping the infant, except for the face, hands, and genito-anal region answers very well. To the genital region and anus an easily changed napkin of cotton or gauze combination may be applied. A jacket may be placed on the outside of the cotton to hold it in place.

Treatment of the Cord.—The time of tying and section of the cord will depend entirely on the general condition of the infant and to some extent on the obstetrician's ability to prevent undue exposure of the infant to cold. In the absence of marked asphyxia it is well to allow the pulsation of the cord to become weakened or to disappear before ligation. This usually requires from one to five minutes during which time the infant will receive from 30 to 60 c.c. of blood from the placenta through uterine contractions and respiration on the part of the infant. This blood should be conserved, when possible, as it is of great value to the premature infant, for its oxygen content and other constituents and nutritive value.

Asphyxia.—The possibility of asphyxiation of the premature infant must be borne in mind throughout the entire labor. The heart tones should be carefully watched and in cases of prolapse of the cord, if it cannot be successfully replaced, it may be necessary to induce a rapid delivery of the infant. Any accumulated secretions of aspirated material must be removed by inversion of the child and if necessary by aspiration with a tracheal catheter. In more extreme degrees of asphyxia early separation of the cord may be necessary so that artificial respiration and a hot bath may be instituted.

All premature infants whether asphyxiated at birth or not should be carefully watched for cyanotic attacks during the first days of life, as such attacks may develop suddenly and without warning. They may be due to a disturbance in the pulmonary circulation, to a congenital atelectasis, or to injury or hemorrhage into the respiratory center in the medulla. At other times attacks of cyanosis are precipitated by intra-abdominal distention which may interfere with cardiac or respiratory action.

GENERAL HYGIENE AND ENVIRONMENT

Requirements of a Hospital Nursery Unit.—This depends greatly upon the method used for maintaining external heat.

1. Superheated rooms without heated beds.
2. Individual heated beds.

When the *superheated rooms* are in use, separate rooms for the older and better developed infants must be supplied to gradually accustom them to ordinary temperature. However, this extra room is not necessary when external heat is applied in individual beds in which the temperature can be regulated to meet the needs of each infant. In using the latter, the room can be held at a temperature approximating 75°F. In point of economy of space and special care for the infant, the latter method has every advantage.

When individual heated beds are used, the following units are required in a properly regulated department.

Room Containing Heated Bed.—In such a room the matter of ventilation will depend to a large extent upon the type of heated bed which is used. When the old type of closed incubator is used, it must necessarily receive fresh air through a pipe passing through the wall of the building or an opening in the window, thereby supplying the bed with air from the outside. When an electrically heated bed or home improvised bed is used, the infant is dependent upon the general ventilation of the room for its supply of fresh air.

Such a room is best constructed with double windows and transom which can be regulated at will according to the season and existing weather conditions. The system of ventilation should be sufficiently flexible to allow of regulation to meet exigencies which may arise due to instability of the general heating plant. It has been our experience that when a well constructed superheated bed is used, variations of from 6 to 8°F. in the room temperature during the 24 hours cause little inconvenience to the infant.

Aseptic nursing is imperative to the welfare of the department. Soiled linens, clothes, bottles, thermometers, and all other utensils must be handled as infected material.

A *complete department* should therefore provide for:

(*A*) *Well Infants.*—A room containing heated beds for the early care and cribs for those infants who no longer need incubator care. The further needs are: A heated dressing table; a supply closet; thermometer (high and low); hygrometer; time clock; electric heater for emergency; screens; and lavatory. A nursery with bathing facilities, supplied with: a bath slab; a lavatory; a heated dressing table; shelves for toilet articles; a gas or electric plate; an electric heater for emergency; a scale; thermometer; supply closet.

A special bath room when possible should be provided so that bathing in the nursery may be avoided.

Quarters for wet nurses with independent bath and toilet facilities, equipped with: Beds, cribs; chiffoniers; dressing table; nursery chairs; and bath room. The bath room should have a shower bath, dressing room, toilet and lavatory.

A milk station containing: a sink; refrigerator; work-table; tubs for washing utensils; steam sterilizer; bottle and food racks.

Nursing staff including a directing nurse and assistants.

Wet nurses.

(*B*) *Infected Infants.*—Room equipped with heated beds and cribs and provided with bathing facilities. This room should further contain a lavatory, heated dressing table, scale, thermometer, hygrometer, emergency electric heater, supply closet, and screens. The bath tub in this room may be of the small ambulatory type.

The nursery should be considered as the center of the unit and when a separate bath room is provided, the former may be used for housing the more vigorous infants. The temperature of this room should range between 78 and 80°F. during the hour of bathing, at other times 70 to 75°F. The entire station must be thoroughly cleaned at least every second day and disinfected by scrubbing immediately after the diagnosis and removal of infectious cases.

The Nursery Staff.—The selection of a personnel for the nursing staff of a unit established for the care of premature infants requires great care. Nurses assuming these responsibilities must be intensely interested in their work. They must be willing to make many necessary sacrifices while the infant is passing through the critical stages. They must, at all times, be prepared to meet the emergencies of asphyxia and to counteract the spells of cyanosis. These two factors in themselves require almost constant diligence, otherwise the work of previous days will go unrewarded. They must use good judgment to prevent over- and underfeeding, as to a very great extent the size of the individual meal will be dependent upon the physical condition of the infant at the time of feeding. In no other class of patients is it so necessary to change or modify on short notice previous orders for diet. The nurse must know the indications for and the methods of administering catheter feedings, colonic flushing, tubbing, and the application of artificial respiration.

In our hospital wards we have found the constant changing of nurses, as is so frequently the case in meeting the curriculum for nurses' training in general hospitals, to be of the greatest disadvantage. Far better results are obtained when the nurse in charge has under her care assistants who need not necessarily be nurses in training, but preferably young women who are especially preparing themselves for the care of young infants, and who can be relied upon to stay in the station for long periods of time. Such women become expert in the handling of these infants, can frequently feed them with a minimum of excitement of their reflexes, and soon learn to bathe and give them their exercise and massage, which is so essential to every infant in order to prevent "hospitalization."

The *ideal nursing staff* for such a station is, therefore, one consisting of a well-trained supervising nurse and a corps of assistants desiring this training, and who are willing to remain in this service for a long period of time.

Daily Routine.—*The body temperature* must be taken through the rectum. It should be recorded morning and evening. An individual thermometer should be furnished for each infant. Fluctuations in body temperature are more marked than in the full-term infant with a tendency toward *hypothermia.* A minimum of 97°F. should be considered the lowest compatible with progress. Attempts should be made to limit the daily fluctuations to 1½°F.

Subnormal temperature may result from undue exposure at birth, subsequent carelessness, lack of development of the nervous system, absence of a sufficient layer of subcutaneous fat, respiratory insufficiency, circulatory weakness, and insufficient heat production due to lack of food or improper metabolism.

These etiologic factors are to be counteracted by definite therapeutic measures.

Prevent undue exposure and trauma from the moment of birth.

The infant should be placed in a heated bed of proper construction and kept there under constant supervision. The temperature of the heated bed should be varied with the needs of the individual infant. Small prematures and congenital weaklings with marked hypothermia should temporarily have a surrounding temperature varying from

85 to 95°F. Older and stronger infants are better placed in a bed at 75 to 80°F. As the infant develops its vital functions and the subcutaneous fat increases, the temperature of the bed should be gradually lowered to that of the nursery, which should be kept at about 70 to 75°F. It should be the rule to regulate the temperature of the heated bed by the rectal temperature curve, and while it may be impossible to bring the body temperature to normal, the degree of hypothermia is our best guide in the application of external heat.

It may be necessary to place the infant in a hot bath to raise the temperature and stimulate respiratory and cardiac function following syncope.

Removal from the bed should follow definite indications, changing napkins, and the ordinary routine measures can be carried out in the bed. Feeding should preferably be done on a heated table.

The body must be insulated by proper clothing which will be described.

The body fluids, after the first few days, must be maintained by an intake of from one-sixth to one-eighth of the body weight in fluids in 24 hours, and this must include a caloric intake of more than a sustaining diet, 70 calories per kg. after the first few days of life (page 477).

Respiratory and circulatory functions must be protected and at times stimulated.

Hyperpyrexia frequently results from an overheating of the bed, and when a high temperature is noted the temperature of the bed should be considered as a possible cause.

Infections of all kinds tend to the development of fever, but on the whole the reaction is less than in the full-term, however, the exception may be true. We have found massive pneumonias at autopsy which were unassociated with temperature above the average normal. It should be remembered that the infection existed in an individual with a subnormal temperature in whom two to three degrees of fever would bring it up to the normal range.

Weighing should be done with a good scale at a specified time each day as part of the general routine. The infant should, unless contraindications exist, be undressed for this purpose, and this is best done before the bath. The relation between the time of the last feeding and urination and passing of feces should be noted. As the infant usually urinates after or between feedings and the bath is given on an empty stomach, so-called, these factors remain of little importance from day to day.

While in older well infants daily weighing may not be indicated, in prematures it should be done as a routine, and more especially in difficult feeding cases. Those fed at the breast must be weighed before and after nursing, and the food taken is to be recorded.

Loss of body-weight during the first days of life occurs almost constantly in premature infants, the percentage loss being greater in the premature than in the full-term infant, and, on the whole, they are much slower in regaining their birth weight. In the group of cases studied by me the average loss in the cases weighing between 1000 and 2000 gm. was 10.9 per cent. More recently we have occasionally been able to prevent an initial loss and more frequently to limit it to 5 per cent. by pushing the fluid intake after the first 12 hours.

Most of our cases have regained their birth weight by the eighteenth to the twenty-first day, with a daily gain averaging from 12 to 40 gm. after reaching their lowest weight, which is usually about the fifth day. Infants under 1500 gm. may be considered as growing satisfactorily on an average of from 10 to 20 gm., and doubling their birth weight in 75 to 100 days; and those from 1500 to 2000 gm. when they are making a daily gain of from 15 to 30 gm. after they have reached or passed their birth weight with a doubling of birth weight in from 50 to 100 days.

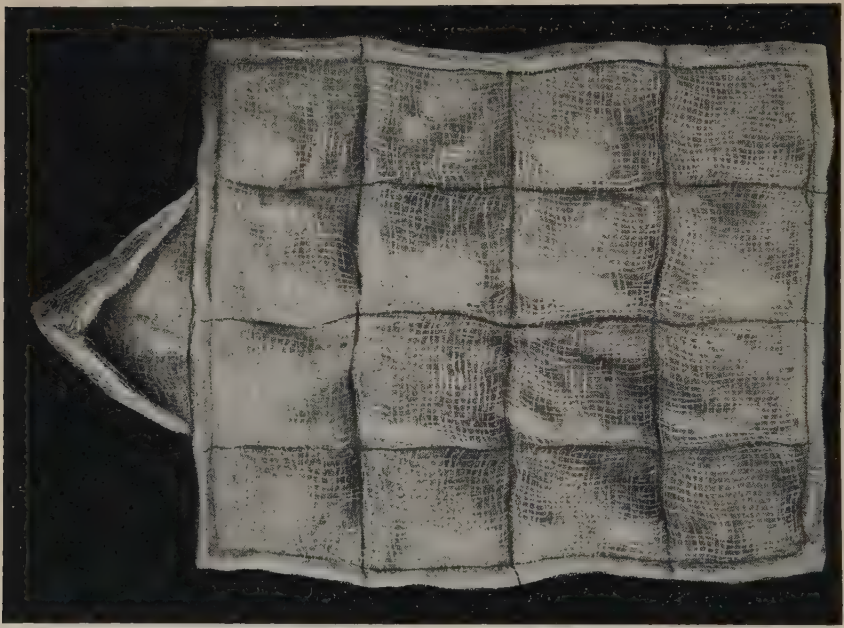


Fig. 119.—Emergency robe with hood made of gauze and cotton combination.

The Infant's Clothes.—The wardrobe should be planned and completed in advance of labor. In emergencies this may not be possible. It is imperative to remember that preservation of the body heat must be begun immediately after birth; on the confinement bed itself. Insulation of the body is the prime thought to be borne in mind when planning the wardrobe. The clothes must fit the body snugly, providing only for a thin layer of air between the body and the dress. The material must be selected with some knowledge of the method by which external heat is to be supplied. The head, except the face, and the extremities must be equally protected with the body.

At birth the infant is received into a warm blanket and immediately placed in a heated basket, heated bed, or incubator. In small prematures for temporary emergency use, a sterile cotton-pack which completely envelopes the infant, except for the face and genito-anal region, may be applied. It should, however, be remembered that cotton is far inferior to wool in prevention of heat radiation. To the genital region and anus an easily changed small napkin of cotton or gauze combination may be applied. An improvised jacket, preferably of flannel, may be placed on the outside of the cotton to hold it in place. Whenever the infant becomes soiled, it is only necessary to change the napkin. This should not be neglected.

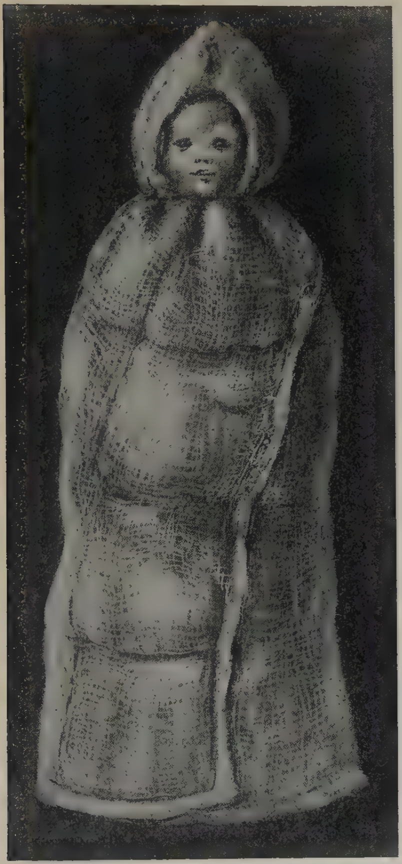


Fig. 120.—Emergency robe applied to infant.

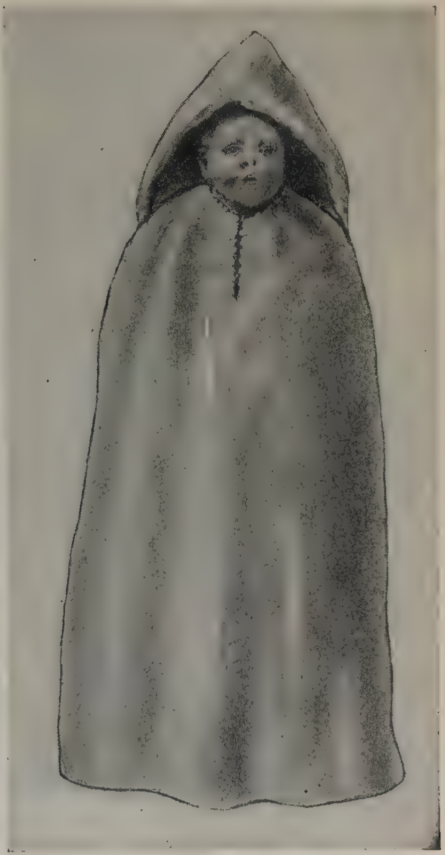


Fig. 121.—Woolen bag with hood. For further protection it may be drawn together beneath the infant's chin.

If special outer garments are not available, the infant should at once be wrapped in a small heavy woolen blanket, or cotton combination (Figs. 119, 120), which can be fastened about the body loosely by bandages or safety-pins in papoose fashion. The greatest disadvantage of such a dress is the limitation of body movements, which is of considerable importance even in these infants. All pressure and constriction must be avoided.

In a well-equipped station several sets of special cloths should be provided. These should be kept sterilized in packets. The outfits will differ some, depending upon whether the open or closed incubator beds are used.

With the *open type of heated beds*, all garments next to the body, except the napkins, should be made of light-weight flannel.

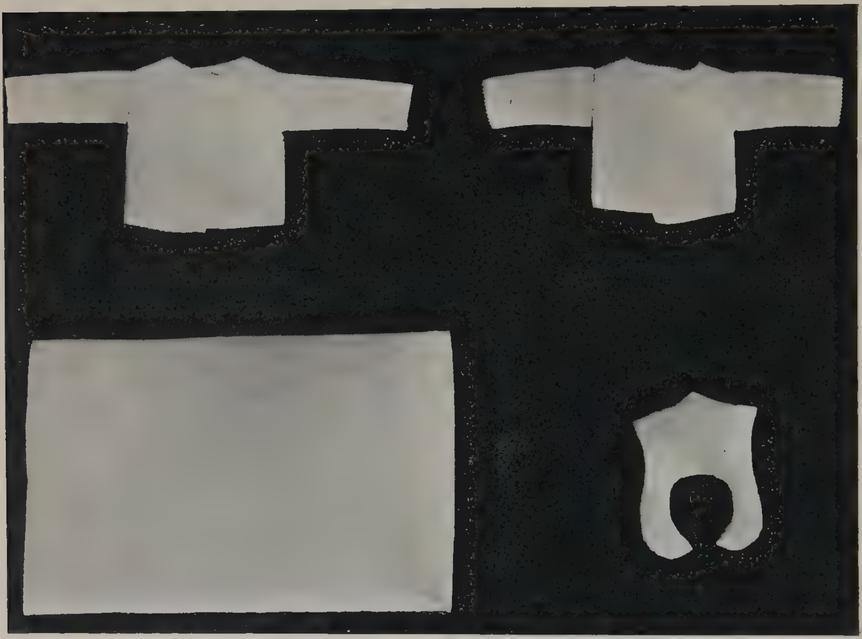


Fig. 122.—Set of clothes for premature infants. Undershirt—flannel with blind sleeves. Overshirt—French pique. Pinning skirt—French pique (24–48 inches). Bib. Further needs which are not illustrated—diapers (18 by 20 inches), woolen stockings and bag or blanket.

A set of clothing should consist of woolen bands of small size; small woolen undershirts; overshirts; pinning skirts; woolen stockings; diapers; pads; bibs; and a woolen bag, with an attached head-piece, with a slit over the upper part in front to allow passing over the head. The bag should be open at the bottom so that it may be raised for changing of napkins, dressing the cord, and general care of the infant (Figs. 121–124). The overshirt should be somewhat longer and larger than the undershirt and may to very good advantage be made from French pique which is less impervious to air than flannel. The undershirt should have closed sleeves.

In the absence of a sleeping bag, the infants may be wrapped in a light flannel blanket, so applied that the upper part will form a hood.

With the *closed type of bed*, the sleeping bag and blanket are unnecessary.

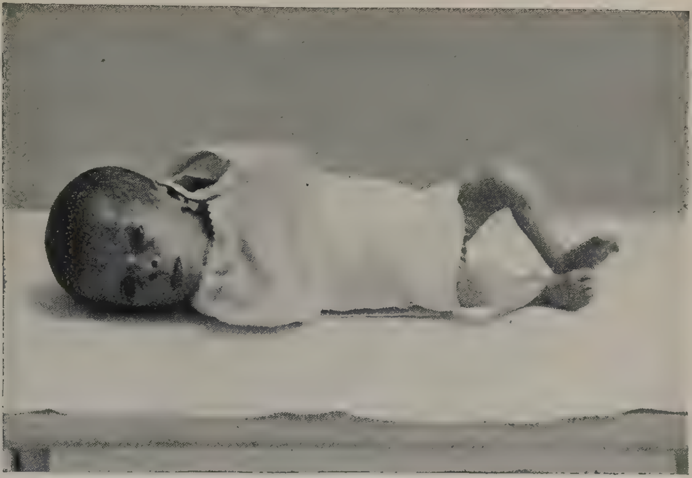


FIG. 123.—Dressing the baby. Under- and overshirts applied.

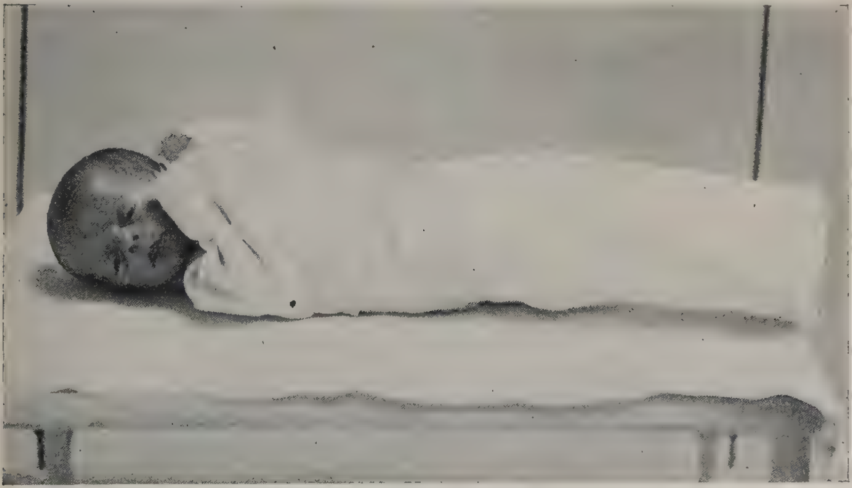
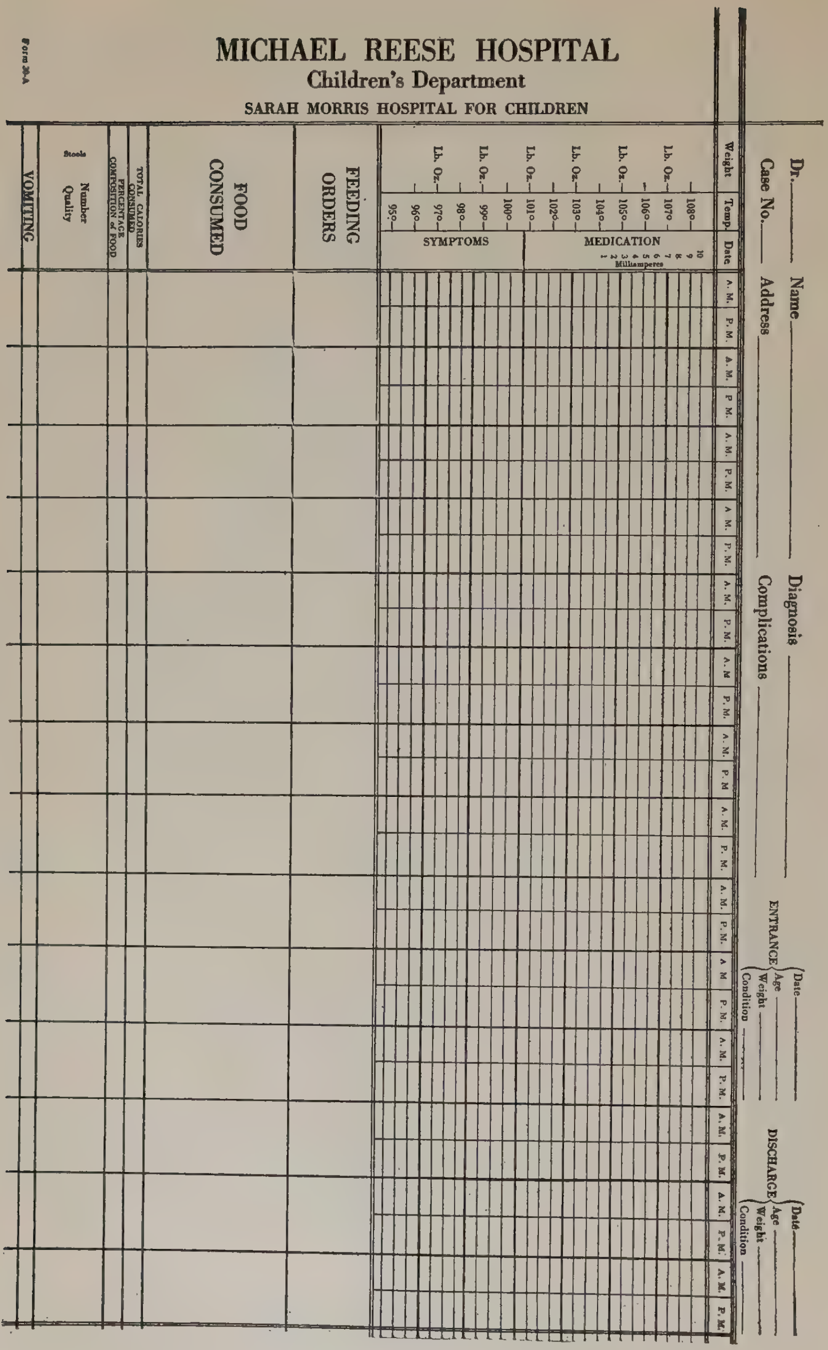


FIG. 124.—Dressing the baby. Under- and overshirts, diaper, stockings and pinning skirt.

Watch for Sickness.—The possibility of grave pathologic changes with minor clinical manifestations must be constantly borne in mind in the care of prematures. In order to diagnose and properly counteract the dangers which may follow the overlooked simple ailments, at least one daily general inspection and examination, quickly but care-



Form 20-A

MICHAEL REESE HOSPITAL
Children's Department
SARAH MORRIS HOSPITAL FOR CHILDREN

Dr.______ Name______ Diagnosis ______

Case No.______ Address______ Complications ______

ENTRANCE Date______ Age ______ Weight ______ Condition ______

DISCHARGE Date______ Age ______ Weight ______ Condition ______

Weight: Lb. Oz. | Lb. Oz. | Lb. Oz. | Lb. Oz. | Lb. Oz. | Lb. Oz.

Temp.: 95° | 96° | 97° | 98° | 99° | 100° | 101° | 102° | 103° | 104° | 105° | 106° | 107° | 108°

Date: A. M. | P. M. | A. M. | P. M. | A. M. | P. M. | A. M. | P. M. | A. M. | P. M. | A. M. | P. M. | A. M. | P. M. | A. M. | P. M. | A. M. | P. M. | A. M. | P. M. | A. M. | P. M. | A. M. | P. M. | A. M. | P. M. | A. M. | P. M.

SYMPTOMS

MEDICATION
1 2 3 4 5 6 7 8 9 10
Milliamperes

FEEDING ORDERS

FOOD CONSUMED

TOTAL CALORIES CONSUMED

PERCENTAGE COMPOSITION of FOOD

Stools
Number
Quality

VOMITING

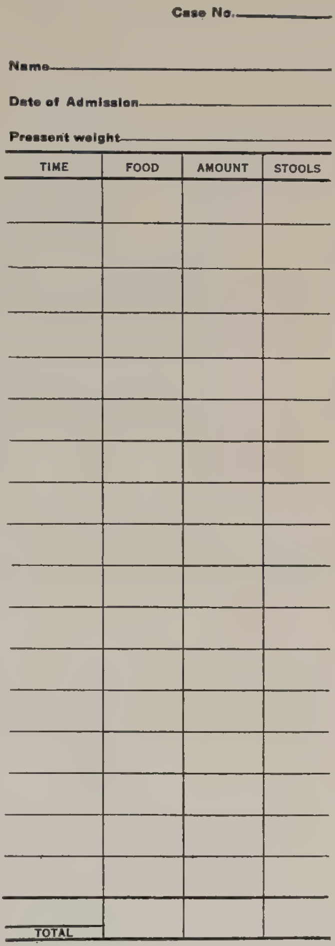
FIG. 125.

fully performed, is required. The exception to this rule is the immediate danger due to handling extremely delicate infants.

In no other group of infants is a careful study of the individual functioning of the heat centers and the respiratory, circulatory, nervous, genito-urinary, and gastro-intestinal organs so imperative.

The Hospital Records.—The records should include the following forms:

1. A history and physical examination blank.
2. A graphic record chart (Fig. 125).
3. A special feeding card for recording the amount of individual feedings and stamped by the time clock. Time of urination and stools and a description of the latter can be recorded on this same card. The latter will avoid neglect in changing the infant and assist in the prevention of local and ascending bladder infections. The data from the feeding card should be transposed to the graphic record sheet daily (Fig. 126).
4. Temperature chart for room and bed. On this sheet is recorded the temperature of the bed in which the baby is kept. It should be charted at six-hour intervals, best at 6 a.m., 12 m., and 6 and 12 p.m. These are the most likely times for maximum changes in the ward temperature which might call for an increase or decrease in the external heat to be applied to meet the desired bed temperature. At the same time, the ward temperature should be recorded. And once daily the humidity in the room and bed should be tested and recorded (Fig. 127).
5. Physician's order blank (Fig. 128).
6. Milk station order blank (Fig. 129).
7. Wet nurse's record blank (Fig. 130).



Case No.________

Name________

Date of Admission________

Present weight________

TIME	FOOD	AMOUNT	STOOLS
TOTAL			

FIG. 126.—Individual diet record sheet.

FIG. 125.—A brief description of the clinical sheet used in our wards may be of value as it answers both the needs of a history sheet and of a daily chart as well. The points illustrated by it are, a graphic relationship between the temperature, weight, quality and quantity of food taken, and the end-results on the stools and urine. Also separate spaces are provided for complications which may influence the preceding under the heading of symptoms, together with spaces for treatment other than dietetic, energy value of foods, vomiting, blood examinations, tuberculin reactions, etc. The small figures 1–10 are used to make an electric reaction curve in cases showing a spasmodic diathesis.

Requirements for the Care of Premature Infants in the Home.—The establishment and maintenance of properly equipped hospital stations are essential to the lowering of mortality, more especially in the large cities and particularly among the poorer classes. A careful consideration of the requirements for and results to be expected from their care in the home is equally essential.

In many instances the premature is born unexpectedly with little time for preparation for its reception. The expectancy of a premature labor is almost always associated with more or less excitement in which thought for the baby's needs are likely to be overlooked, the mother usually being given first consideration.

TEMPERATURE CHART FOR HEATED BED

Name______________________ Dr.______________________
Day of Entrance______________________ Age__________ Temperature__________
Birth weight__________ Present weight__________ Case No.__________

	6 a. m.			12 noon			6 p. m.			12 p. m.		
DATE	Room	Contact	Bed	Room	Contact	Bed	Room	Contact	Bed	Room	Contact	Bed

FIG. 127.

In the home care of these infants the same rules for hygienic maintenance of body temperature, breast feeding and daily routine must be maintained as suggested for their hospital care.

The Nursery Unit.—Whenever possible two rooms should be set aside for the infant's use; one equipped as a nursery with furnishings similar to those described for the hospital nursery. The second room is to be used for sleeping quarters and must be equipped with a heated bed. These rooms must be well ventilated and at the same time well heated. In both these rooms all draperies and unnecessary furniture must be removed.

PREMATURE INFANT STATION

FEEDING ORDERS

DATE	NAME	AGE	WEIGHT	BREAST MILK	NUMBER	AMOUNT	WATER	OTHER FOOD

FIG. 128.—Diet order sheet to be filled in by physician during morning rounds. Day's food orders for premature cases on the service.

PREMATURE INFANT STATION

FOOD REQUIREMENTS FROM MILK STATION

DATE	NAME	AGE	WEIGHT	BREAST MILK	NUMBER	AMOUNT	WATER	OTHER FOOD

FIG. 129.—Duplicate of diet order sheet, Fig. 128. To be filled in by nurse in charge of ward and sent to the wet nurse and milk stations.

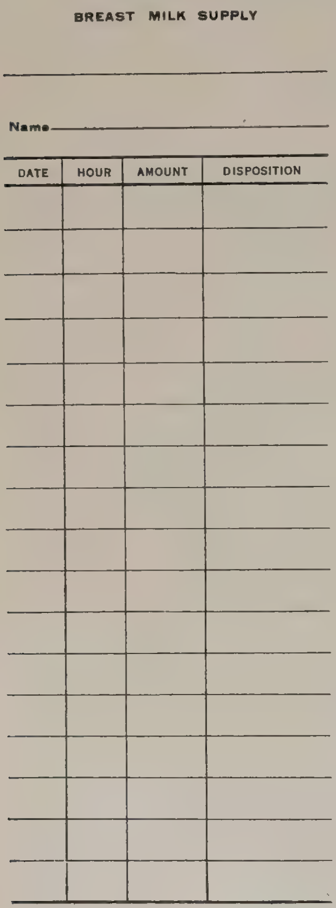
A bath room properly equipped makes a splendid second room in which the general care of the infant can be administered. This room should be given over to the exclusive use of the infant. When such a bath room is available only one other room need be supplied.

While the baby is being dressed or bathed the nursery or bath room temperature should be in the neighborhood of 80°F. A gas or electric stove will be of assistance in accomplishing this. When a superheated bed is in use, the sleeping room may be kept between 70 and 75°F. These rooms should be devoid of all excessive furniture and draperies. *All visitors other than the attendants and physician must be excluded.*

The equipment of the room or rooms should include a heated bed, a dressing table, preferably heated, or placed over a warm radiator, a small electric or gas stove for emergency use, a scale, bathing and feeding utensils, a thermometer, a hygrometer and surgical supplies.

The Superheated Bed.—In the home hot water bottles, a properly protected electric pad or an improvised incubator will answer the purpose (page 486).

A thermometer should be placed alongside the baby as too great emphasis cannot be laid on the dangers and fatalities due to overheating and burning of prematures. There is a great tendency to hyperthermia which must be recognized and properly interpreted. There is usually a rapid return to the normal body temperature without bad effects upon removal of the cause unless too long continued.



BREAST MILK SUPPLY

Name

DATE	HOUR	AMOUNT	DISPOSITION

FIG. 130.—Wet nurse milk supply record sheet. A daily check on the wet nurses. They should be kept on file.

METHODS OF FEEDING

Infants Nursing at the Breast.—This presupposes that the infant has the proper physical development to withdraw milk from the human breast in the presence of an abundant supply and well-developed nipples. Such an infant may be placed on the breast two or three times during the last half of the first day after the circulatory and respiratory functions are well established. Following the first day it should be placed at the mother's breast regularly for two or three-minute periods at three or four-hour intervals, even though the breast contains little milk. Following these attempts at nursing, food should be supplied from another mother or a wet-nurse, whenever such a supply is obtainable rather than to institute artificial feeding. In the hospital it is our custom to give these additional feedings by hand; in private practice the infant may be placed to the wet-nurse's breast, the same breast being used at each nursing for this purpose, and if there is a difference in the breasts, the better one should be selected for the premature. However it is to be remembered that congenital syphilis is to be excluded, both in the infant and wet-nurse, in all cases where the infant is put directly to a breast other than the mother's. Not infrequently great assistance may be given the infant in securing its milk by one of two methods: Either by expressing milk directly into the baby's mouth, or by placing the wet-nurse's baby on the opposite breast which reflexly stimulates the flow of milk into the opposite

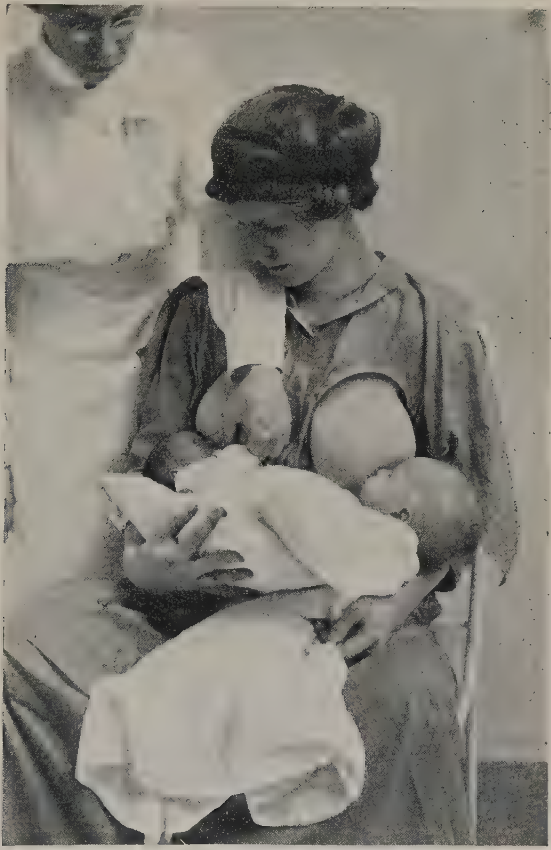


Fig. 131.—Feeding premature infant by direct expression from right breast. Wet nurse baby on left breast assists in stimulating flow of milk into right breast.

breast (Fig. 131), thereby assisting the weak infant in obtaining its food. *Overfeeding* becomes a danger in this direct application of the infant to the breast, and weighing before and after nursing should be practiced. Whenever possible this is the best method of getting the food to the baby, as it prevents contamination of the milk, stimulates the breasts, and develops the baby's independence as well as his sucking muscles. *Underfeeding* is an even greater danger, and here again the infant must be weighed before and after feeding to ascertain the amount of food taken. If insufficient, additional food can be supplied by hand feeding.

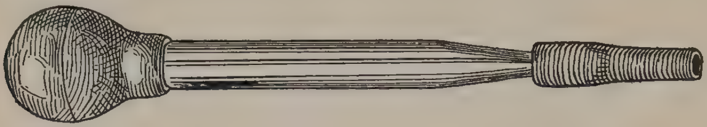


FIG. 132.—Large medicine dropper with a short piece of soft rubber tubing over lower end to prevent injury to the baby's mouth. Most infants soon learn to suckle on the soft rubber tube when inserted into the mouth.

When sufficiently developed a small bottle and nipple can be substituted.

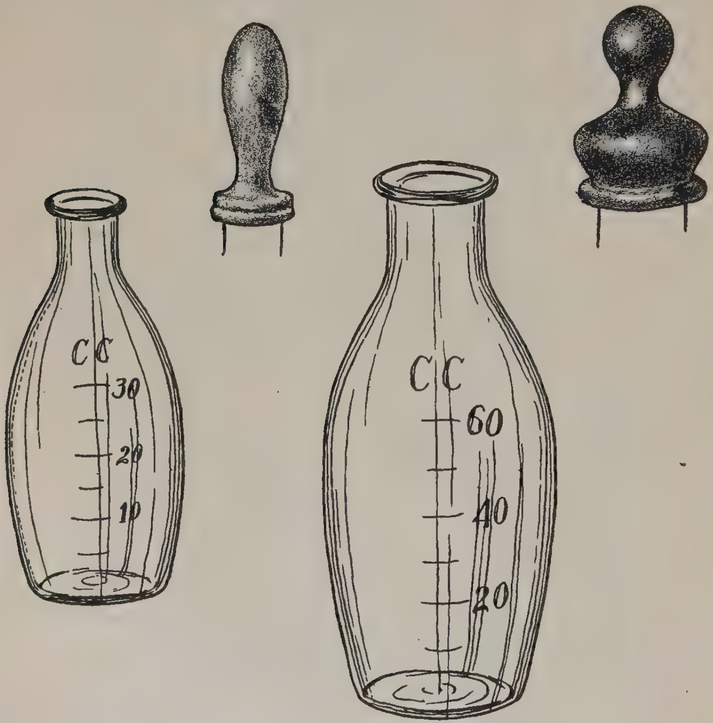


FIG. 133.—One-ounce graduated nursing bottle with small nipple approximately the size of the end of an ordinary medicine dropper.

Two-ounce graduated bottle with a special nipple with a small mouth piece. This nipple will fit on the larger neck bottle sold on the market and can also be inverted for cleansing. Another good nipple is that shown in type two Breck feeder.

Infants Too Weak to Nurse at the Breast.—In this group of infants careless exposure must be avoided, and this is best accomplished so far as possible by feeding them without removal from the bed. Difficult feeding cases and those to be fed by catheter must be placed on a warmed table for feedings. The inability to nurse may be due to improper development of the nursing center, or lack of coördination on the part of the pharyngeal muscles and tongue. The latter is usually made evident by a return flow of milk from the mouth. Again, the infant may be too weak to nurse, or it may not have learned to suck, or vomiting, and again cyanosis may prevent its feeding properly. In this group of infants we may, of necessity, resort to one of several procedures:

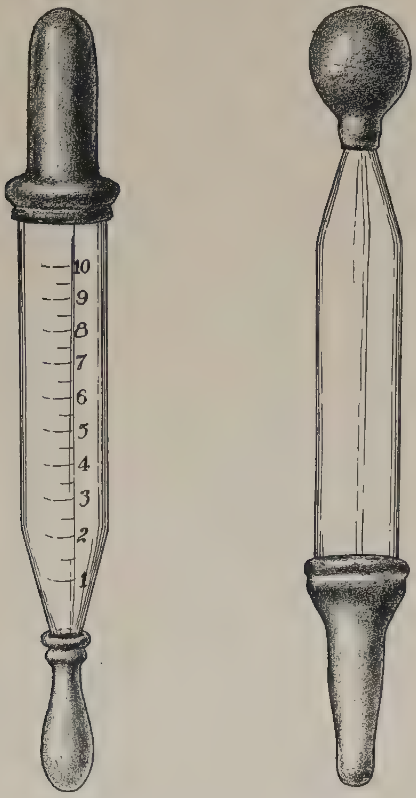


Fig. 134.—Modified Breck feeders.

Type one: With a small nipple at the lower end and an ordinary finger cot at the upper end.

Type two: Has a large nipple at the lower end and a medicine dropper bulb at the upper end. The latter is not a safe model because the milk must be poured into the large end and therefore enters the bulb which is difficult to clean.

A third type can be made by flanging a straight piece of large tubing and using the large nipple at one end and the finger cot at the other. The glass part can be blown by any specialty glass company or the barrel of an ordinary glass syringe can be drawn and flanged to take the rubber parts.

The use of a large size medicine-dropper (Fig. 132) with a short piece of soft rubber tubing passed over the end.

In those infants who can assist themselves:

A small *nursing bottle* (Fig. 133, 1- and 2-oz. bottles). The 1-oz. bottle is provided with small nipples the size of those commonly sold on doll nursing bottles. Such a nipple can be made by perforating the rubber bulb of a better quality medicine-dropper. Our 2-oz. bottle has a larger neck which takes the ordinary size nipple. One with a small mouth piece must be used. The nipple should be made of a soft elastic rubber, and of these the semi-transparent nipples usually answer best.

The Breck feeder in the original, or a modification which can be made by flanging the ends of a urethral syringe, using a heavy rubber finger-cot at one end and a small nipple or perforated soft medicine-dropper tip on the other, will usually suffice. The first type illustrated has the disadvantage of having the milk enter the bulb on filling from the large end. The bulbs are difficult to clean (Fig. 134).

Direct expression of milk into the infant's mouth has proved one of the most valuable expedients in our hands as a method of teaching the infant the act of nursing.

Catheter feeding is the simplest and best method of procedure in the smaller infants, if carefully practiced by an experienced nurse. Catheter feeding should be instituted as soon as fatigue or cyanosis is noted following other methods of feeding. A catheter (No. 12 French, No. 8 American, No. 5 English) about 14 in. in length may be attached to a small funnel, graduated glass tube, or, in case of emergency, the glass barrel of a small syringe may be used. All food should be carefully measured and administered slowly with a *minimum elevation* required to obtain a free flow of the milk. The infant should be upon its back on a flat surface with the head either in the median line or turned to the right. The passage of the catheter is usually effected without difficulty by passing it in the midline to the pharynx, gradually pushing it into the esophagus. The poorly developed reflexes rarely result in retching. The distance to which the catheter is to be passed is of great importance when we consider that this procedure must be repeated at least six to eight times daily over a considerable period of time. It has been our rule to measure the distance from the bridge of the nose to the tip of the ensiform cartilage, which is usually in the neighborhood of 12 to 15 cm. (Full-term newborn infants average about 16 cm.) The catheter is marked at this point with indelible ink and is passed to this point or about 1 cm. further than this distance which allows it to reach the lower end of the esophagus just above the cardia, from which point the food will flow through the patent cardia. We thereby avoid irritating the gastric mucosa and stimulation of the reflexes at the cardia. One soon learns the distance the catheter can be passed in each case in order to avoid retching. The milk is now allowed to flow into the stomach slowly, the funnel being raised only slightly above the level of the body, usually 6 or

8 in. will suffice. After the feeding the catheter is firmly compressed to avoid spilling milk into the pharynx during its removal. The infant should be turned on its right side following the feeding. In the presence of gastric distention, raising the infant before and after feeding to the vertical position, avoiding flexion of the body, will allow of the eructation of air and frequently prevent cyanosis. When the stomach is noticeably distended with gas before feeding, the catheter should be passed 1 or 2 cm. further than the mark on the catheter before starting feeding, in order to allow the gas to escape. It is then retracted as directed and feeding started. The catheter should be passed with the funnel empty, so as to allow of this procedure. The catheter should then be compressed and the milk poured into the glass funnel. This allows the air in the funnel to escape thereby preventing over-distention of the stomach by the mixture of food and air. The feeding period should be as short as possible without undue haste. Too rapid feeding is more dangerous than too slow. Usually, one to three minutes are needed. Two nurses can be used to advantage in catheter feeding, but, as so frequently happens, only one is available during the night feedings. Every nurse should be trained to under-

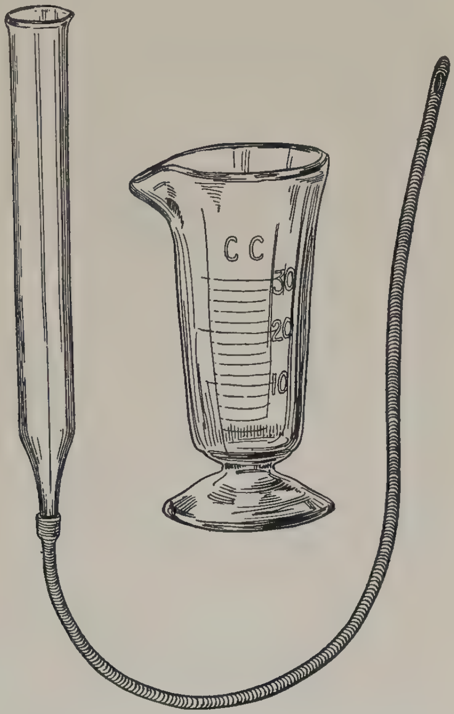


FIG. 135.—Utensils for catheter feeding.

Glass barrel of syringe, No. 12 French catheter and 1-oz. graduate glass. The catheter should be marked at 2-cm. intervals between the distances 12 to 20 cm. above the tip.

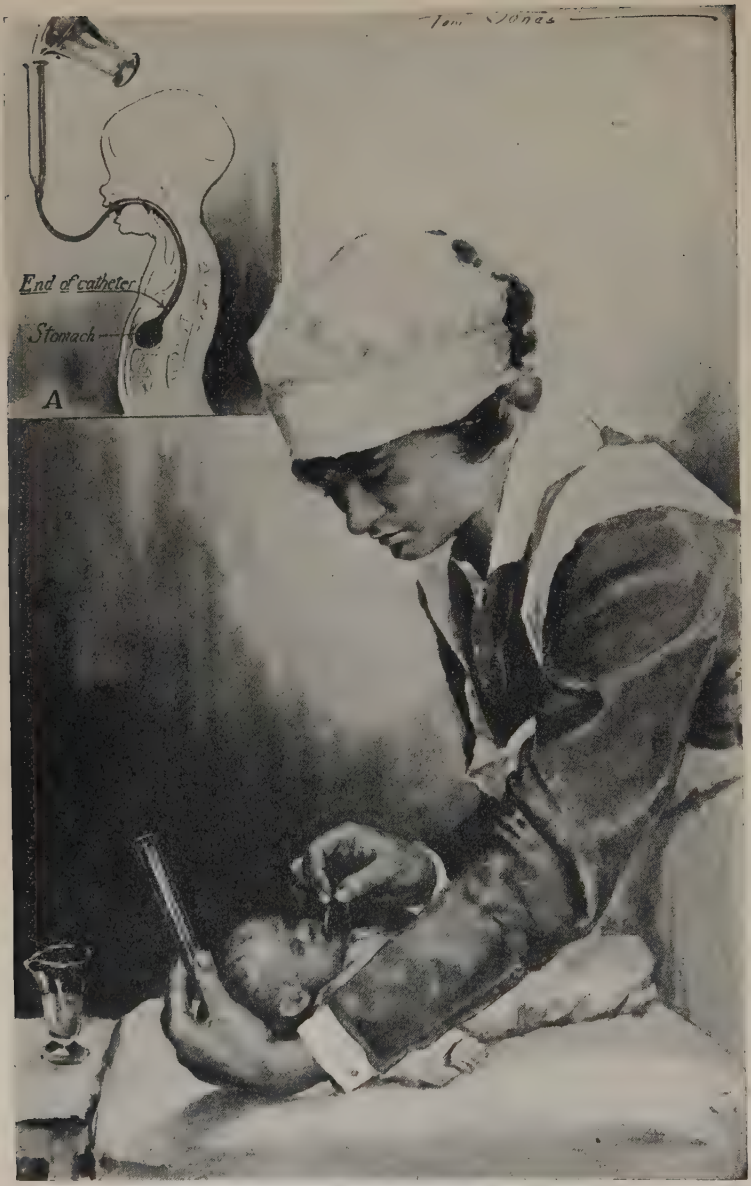


FIG. 136.—Catheter feeding as carried out by one person. The head is held at right angles by the left hand, the catheter is passed with the right hand. Next the funnel is passed to the left hand and elevated to allow the air to escape from the stomach. The catheter is now compressed and slightly elevated and part or all of the feeding is poured in from the graduate and allowed to slowly flow into the stomach.

The small sketch illustrates the point to which the lower end of the catheter should be passed.

take catheter feedings without assistance. We believe that turning the infant on the right side following feedings shortens the emptying time. Its position should be changed at least once between feedings to avoid localized pulmonary congestion (Figs. 135, 136).

The Number of Feedings.—This will, of necessity, depend in many instances upon the question of catheter versus other methods of feeding.* Larger infants fed by catheter can often be given sufficient food at four-hour intervals to meet their needs. In small infants fed by a dropper, bottle, or other methods we have experienced great difficulty in administering a sufficient quantity of food by the long-interval feeding. As the attendants in charge are frequently not to be trusted with the catheter feeding, the short-interval feeding must be resorted to.

For this purpose we have grouped our infants into two classes, those weighing under 1500 gm. and those weighing above this figure. These figures are arbitrary and will not require rigid adherence. The classification is based on the tendency of the smaller infants to become exhausted when the feedings are too long continued. The smaller hand-fed infants are fed at two-hour intervals during the day and three hours at night. The larger on the three-hour basis. When catheter feeding is the method of choice even in the smaller infants six to eight is usually the maximum number needed in 24 hours. It must be remembered that all feedings are dependent on the general development of the infant in relation to its digestion and metabolism and its ability to retain the food administered, as well as attendant complications to feeding, such as asphyxia, cyanosis, and gastric distention.

When to Start Regular Feeding.—This is a question of great importance to these infants, because of the tendency to develop acute inanition. Therefore, a regular feeding regime must be started early. Human milk is essential to a low mortality. As little can be expected from the mother for several days, it becomes necessary to obtain the limited supply necessary from another mother, preferably a wet-nurse. If for any reason it is unlikely that the mother may be depended upon, either because of illness or local breast conditions, immediate search should be begun for a supply of breast milk.

Feeding During the First Day.—During the *first day* it is our custom to withhold milk for 12 hours until the respiratory and circulatory functions are well established. During the second 12 hours one to three feedings of breast milk may be started if the infant's condition warrants.

Feeding from the Second to the Tenth Day.—The *second to the tenth days* may be grouped together as the second feeding period for practical feeding purposes.

From the second day they should be fed regularly, day and night, the number and time of feedings depending to a great extent on whether the food be given with or without the use of a catheter; second, upon the gastric capacity; third, upon the infant's general condition.

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* As there are no short catheters on the market marked in the Metric System it is advisable to mark several for ward use between 8 and 15 cm.

Additional fluids, preferably inert, such as water or 1 per cent. lactose solution, are administered to compensate for the loss of body fluids through the kidneys, bowels, lungs and skin. The infant requires about one-sixth of its body-weight of water, inclusive of that contained in the milk, in 24 hours while in the heated bed. Such quantities, however, should not be attempted on the first days, usually it will be possible to approximate one-eighth of the body-weight by the fourth day. The early feedings must necessarily be small, and the increases gradual.

Each infant must be fed individually, as it is impossible to formulate definite rules for feeding, at least during the first ten days.

First, we must have a definite idea of the minimum food requirements for life.

Second, the amount of food necessary to maintain at least a stationary weight.

Third, the amount of food needed to meet the requirements for growth and development.

Approximately one-seventh of the body-weight of fluids and human milk of a food value of 70 calories per kg. every 24 hours are required to maintain life. Little can be expected in the way of weight increase until 90 calories are reached, and depending on their weight, body surface, and physiologic development, their later needs will approximate 100 to 140 calories per kilogram body-weight.

CALORIC REQUIREMENTS PER KILOGRAM BODY-WEIGHT

Values Recommended by Different Authors

Salge[*]	130–150.
Samelson[†]	115–150.
Oppenheimer[‡]	120–130.
Czerny-Keller[§]	100–120.
Langstein-Meyer[‖]	120–130
Budin[¶]	140 average.
Birk[**]	100–160.
Reiche[††]	120–130 (In those under 2000 gm.) 95–110 (In those over 2000 gm.)
Oberwarth[‡‡]	120–160.
Morse and Talbot[§§]	120 average.
E. Moll[‖‖]	110–120.

[*] Einführing in die moderne Kinderheilkunde, Berlin, 1909.

[†] Zeitschr. f. Kinderh., xi, 1911, p. 18.

[‡] M. K., vi, 92, 1908.

[§] Des Kindes Ernährung usw. Leipsig u. Wien., 1912.

[‖] Säuglingsnarg u. Säuglingsstoffwechsel, Wiesbaden, 1914.

[¶] The Nursling, London, 1907.

[**] Sauglingskrankheiten, 1913.

[††] Z. K., xii, 369, 1914.

[‡‡] Ergeb. d. Inn. Med. u. Kinderh., vii, 191, 1911.

[§§] Diseases of Nutrition and Infant Feeding, New York, 1920.

[‖‖] Ztschrft. f. Kinderh., October, 1919, p. 329.

Infants, to fulfill all their needs, will therefore require from 140 to 200 c.c. of breast milk per kg., or about one-seventh to one-fifth of their body-weight daily. They can, however, maintain life on 100 c.c. and hold their weight in most cases on 130 c.c. per kg.

Beginning (in most cases by the second day) with 20 to 40 c.c. of human milk per kg. of body-weight, the quantity may be increased by 8 to 15 c.c. daily per kg. until, usually by the tenth day, feedings averaging from 80 to 140 c.c. per kg. can be fed.

These feedings should, as rapidly as possible, be supplemented by water or sugar-water by mouth, or saline by rectum to meet the required 140 to 200 c.c. per kg. of fluids required daily.

After the *tenth day* in larger infants the milk can be increased more rapidly, usually by 15 and occasionally 20 c.c. per day, until from 140 to 200 c.c. per kg. are fed, the methods of giving the food, as well as its frequency being dependent on the general development of the infant.

The size of individual feedings will vary with the method of feedings. When *catheter* fed, six to eight feedings a day are given, with an average of from 4 to 6 c.c. per feeding during the second day. The feedings are now increased daily by an average of 2 c.c. per feeding. When feeding from the *bottle* or by *dropper*, smaller feedings are usually given more frequently, usually from 8 to 10 daily, although 12 may be needed when larger feedings are not retained. Begin with 2 to 4 c.c. and increasing by 1 or 2 c.c. per feeding on each succeeding day, until 140 to 200 c.c. per kg. per day are reached.

The food and water to be administered should be noted in writing for the nurse's instruction each day, after a thorough inspection of the infant and its clinical chart.

The diet of a premature infant making a satisfactory gain in weight should not be changed arbitrarily without a well-defined indication.

Initial Weight Loss.—The lower the birth-weight, the greater is the percentage weight loss to be expected. Artificially fed infants lose more weight than breast fed infants in whom the diet is started early. An average loss of not more than 8 to 12 per cent. of the birth-weight may be considered satisfactory. By the regular administration of inert fluids during the first days the total loss can frequently be reduced to 5 per cent.

Daily Gains.—These are not necessarily in proportion to the changing quantity of milk administered, as many factors, such as condition of the bowels, quantity of urine passed, temperature of the infant's surroundings, and numerous other factors will necessarily influence the weight.

An average daily gain greater than 20 gm. is unusual when the infant's food is limited to one-sixth of its body-weight. Although occasionally an infant holds its birth weight, most infants do not regain their birth-weight before the end of the second or third week. In the very small prematures an average daily gain of 10 to 15 gm. with a doubling in birth weight in from 75 to 100 days may be considered satisfactory. In the larger infants a gain of 15 to 20 gm. may be expected with a doubling in birth weight in from 50 to 100 days. The birth-weight is frequently trebled within 180 days.

Special Feeding Rules.—(1) Food requirements which have been recommended must of necessity be considered as relative, variations being to a great extent influenced by the physiological and anatomical developments and to a not inconsiderable extent by the temperature and humidity of the air surrounding the infant and the type of clothes in which it is dressed.

2. Each day the total amount of food as indicated for the individual infant is to be estimated, in order that the required food and water will be properly administered. The number and amount of feedings will of necessity vary, but each must also be estimated every day.

3. When a number of infants are to be fed by one wet-nurse, careful calculation of the day's needs of each infant must be made by the floor nurse for the information of the nurse in charge of the milk supply.

4. Expression of breast milk should be performed at regular intervals preferably six times a day at four-hour periods day and night. The sixth expression during the night may, however, be omitted if the supply is in excess. It is only by regular and complete emptying of the breasts by expression that a milk supply can be maintained for an indefinite period, unless there is a second baby which can be placed at the breast.

5. Human, as well as cow's milk, must be obtained under aseptic conditions and kept clean and cool until ready for the infant. To preserve milk properly, the ice box must register less than 50°F. The food should be slowly warmed before feeding.

6. The amount of water to be fed must be carefully calculated, and it must represent the difference between the total fluids indicated which will usually average from one-eighth to one-fifth of the body-weight of the infant for 24 hours and the amount of fluid given as milk. *The water for each day should be measured and set aside in an individual stoppered bottle each morning.*

It should be administered between the milk meals or occasionally there may be an indication for diluting the milk with part of it. In order to administer the full day's water supply in some of the small infants and those who vomit, it may be necessary to give water in small quantities one, two, and even three times between milk feedings. If unable to swallow properly, water must be given by catheter. In larger infants only a few water feedings a day may be needed, and usually by the third or fourth week, one-seventh or one-fifth of the body-weight in milk can be fed daily. At this time the water may be discontinued unless it is necessary to supply external heat of considerable degree, or the infant has a fever, both of which necessitate increased amount of fluids.

Feedings after the Twenty-first Day.—Usually by the twenty-first day, the food requirements of the infant are quite well established and a careful observation of the infant's weight, stools, disposition, and equally important, its body temperature will decide the further food requirements.

The water requirement will to a great extent be dependent upon the supply of artificial heat and the presence of fever. Ordinarily by the beginning of the fourth week, one-seventh to one-fifth (140 to 200 c.c. or 100 to 140 calories per kg.) of the infant's body-weight in the form of breast milk is needed to maintain proper growth. Rarely is it necessary to exceed these amounts even in the poorly nourished premature. If the physiological functions are seemingly normal, *the scale* is the deciding factor in indicating food increases or decreases.

As the infant takes on weight and becomes fat with a rounding of the features and the body, as is the case in successfully breast milk fed prematures, the total milk administration can be held at one-sixth and not infrequently one-seventh of the body-weight, and normal weight increases may still be maintained.

Mixed Feeding.—When human milk, even though in small quantities, is available, it should form the basis of the diet, and cow's milk mixtures should be supplemental.

Artificial Feeding.—There can be no comparison between the results to be expected in feeding premature infants on human milk and those to be obtained with artificial food. This is especially true of infants with a weight below 1500 gm. Therefore, if it becomes necessary to resort to artificial feeding, the selection of a food, its preparation, and its adaption to the infant must all be given the most painstaking consideration. Many varieties of artificial diet have been suggested by many different authors, such as simple milk dilutions, cream and top-milk mixtures, skimmed milk and buttermilk preparations, malt soup preparations, condensed and evaporated milk, etc. The results with these various diets are to a great degree dependent upon the physician's intimate understanding and directions for the use of the individual food.

Quantity of Food.—It must be remembered that the figures quoted for the feeding on breast milk are the maximum that can be assimilated and are excessive quantities for artificial feeding in the first weeks of life because of greater difficulty in the digestion of cow's milk. These infants when artificially fed must at all times be closely watched for evidences of overfeeding, and the first evidence of digestive disturbances or of intercurrent infections should lead to the feeding of human milk whenever possible.

From the foregoing statement, it is quite evident that smaller and slower weight increase may be expected of the artificially fed.

Quality of Food.—As previously stated, opinions vary greatly as to the best food for artificial diet. Most clinicians have obtained the best results with the feeding of low fat mixtures. Boiled milk, skimmed milk, and buttermilk, with carbohydrates added, are among the best.

In feeding with buttermilk and skim milk with added carbohydrates, the fat free mixtures must not be too long continued, otherwise the infants will suffer from fat inanition. By the end of the third week some whole boiled milk may be added or the lactic acid milk should be prepared from partly skimmed milk. It is our routine to *boil all artificial food mixtures* for at least two minutes.

In feeding with a buttermilk high *carbohydrate mixture*, the caloric requirements are lower than in feeding with the full milk (chymogen) mixture with its *lower carbohydrate* content, because the energy for digestion and assimilation requirements are lower with high carbohydrate low fat feeding as compared with high fat low carbohydrate mixture.

BUTTERMILK AND SKIM MILK MIXTURES

Buttermilk or skim milk	1000
Flour (dextrinized)	10
Sugar (cane)	40

The above being used for the first weeks.

Buttermilk or skim milk	1000
Flour (dextrinized)	15
Sugar (cane)	60

For later feedings.

Maltose-dextrin compounds can be substituted for the cane sugar if desired.

Directions for Preparation

Add the flour to a few tablespoonfuls of buttermilk or skim milk and rub to a paste. Add buttermilk or skim milk to one liter.

Bring mixture to a boil and withdraw from the fire.

Add the sugar and bring to a boil for the second time.

This process should require about 20 minutes.

The mixture should be stirred constantly with an egg beater while over the flame.

Make up to one liter with boiled water.

Keep on ice.

In the use of the buttermilk mixture it must be remembered that infants are not to be kept on this low fat mixture for too long a time, addition of whole milk being indicated after the first few weeks, beginning with one-third whole milk and two-thirds skim; by the fourth week equal parts whole and skim milk should be used in the preparation of the lactic acid milk.

CHYMOGEN MILK

Requirements for preparation
Milk,
Single boiler, 1 or 2 qt.,
Dairy thermometer,
Chymogen powder. (Armour and Co.)
Teaspoon,
Egg beater.

Directions for Preparation.—Boil milk for five minutes over direct flame, cool to 104°F., and add one full teaspoonful of chymogen to each quart of milk, and stir for one-half minute. Let it come to a clabber by allowing it to stand for 15 minutes, holding it as near 100°F. as possible by keeping it in a warm place; then beat it well until the curd is finely divided. Put it in individual feeding bottles and place on ice. Do not heat above 100°F. when preparing individual bottles for feeding, otherwise curds will clump and will not pass through the nipple. Reheating is best accomplished by placing the individual feeding bottle in a cup of warm water not over 110°F. and allowing it to stand for 10 minutes. Replenish the warm water if necessary.

We have found this predigestion of boiled milk, by the addition of rennet (chymogen) assures the infant's stomach of a fine, flocculent curd, which is about the size of that of human milk. In beginning feedings with the above preparation it is usually diluted with three parts of water and increases in quality made as indicated, and the quantity increased as in the feeding of human milk. In feeding with the diluted predigested milk, 15 gm. (½ oz.) of lactose should be added to each liter during the first few days, and the amount gradually increased to 30 gm. (1 oz.). When chymogen is not available, whole milk boiled for five minutes may be used.

In the feeding of these food mixtures, the relative caloric values of the mixtures as compared with breast milk must be borne in mind, otherwise inanition will be the result.

Human milk = 700 per liter or 21 calories per ounce.

Skim milk or buttermilk mixture containing 10 gm. dextrinized flour and 40 gm. sugar per liter = 16 calories per ounce or 525 per liter.

Chymogen milk (whole milk) = 700 per liter or 21 calories per ounce.

Chymogen milk when diluted with three parts water and 15 gm. of sugar per liter = 235 calories per liter or 7 per ounce.

Chymogen milk when diluted with equal parts water and 30 gm. of sugar per liter of mixture = 470 per liter or 14 per ounce.

Each individual ounce of sugar per liter of mixture increases its food value by four calories per ounce. Rarely should more than 2 oz. of sugar be added for each liter of the mixture, which represents an afddition of 6.5 per cent. of carbohydrate to that already contained in the milk. It is, therefore, of the greatest importance that the milk itself either skimmed, buttermilk, or whole be increased gradually along with the sugar.

Amounts to be Fed.—Depending upon the weight and development of the infant, the tables as given for human milk feeding should be followed. It must be remembered that while the artificial diets recommended have a lesser caloric value per milliliter than human milk, they represent for most infants the maximum capacity for digestion and assimilation. Of necessity, lesser weight increases and slower progress are to be expected. The infants are paler, tissue turgor is lacking, and they are less immune to infection.

Other Dietetic Requirements.—To counteract the effects of boiling, orange juice feeding should be instituted by the third week, beginning with 0.5 c.c. (8 drops) and increasing to 2–4 c.c. (1 to 2 dr.) daily by the eighth week, in order to avoid scurvy. Cod-liver oil as an antirachitic should be fed by the fourth week, beginning with 0.5 c.c. (8 drops) daily divided into two feedings and increased to 2 c.c. (30 drops) daily by the eighth week. It may be mixed with the orange juice. To counteract the low iron content of these diets, carbonate of iron in 0.03 gm. (gr. ½) or citrate of iron and ammonia in 0.03 gm. (gr. ½) once daily should be started by the fourth week. The latter may be prescribed in solution.

INCUBATORS

In 1857, Denucé first described his model which consisted of a double walled tub which was to be filled with warm water at intervals. Credé in 1866 published the results which he obtained with a similar apparatus which had been in use in his clinic since 1860. (Fig. 137.)

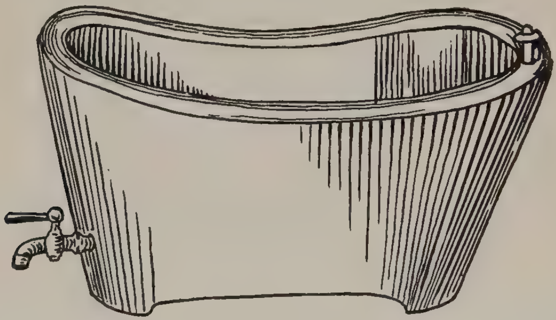

Fig. 137.—Warm tub with double wall jacket. First made by Denucé in 1857 and Credé in 1860.

In 1880, Tarnier had an infant incubator constructed similar to those used as chicken incubators. This is the first closed incubator which may be qualified as modern, for the perfected apparatus of our day differs from it only in detail (Fig. 138).

This is the time that dates the principal work undertaken on incubator construction; and the most varied modifications have followed each other until our day.

The Lion incubator (Fig. 139) was the first attempt at construction of a so-called perfected type with special provisions for filtering the air, hot water heating, constant circulation of air and automatic temperature and humidity control. To this class belong the models of Couney, DeLee and others on the market in the United States.

These models differ but slightly in principle, the chief variation being in the manner of heating and distributing the air and supplying moisture. They may be heated by gas or oil stoves situated at the side of the incubator, heating the air as it enters, or by a system of electric bulbs within the incubator. In the latter models the bulbs are usually located either in the floor or sides. The best models are those in which the heating system is modeled after that used in hot water heating plants for houses. The temperature is automatically controlled by a thermostat.

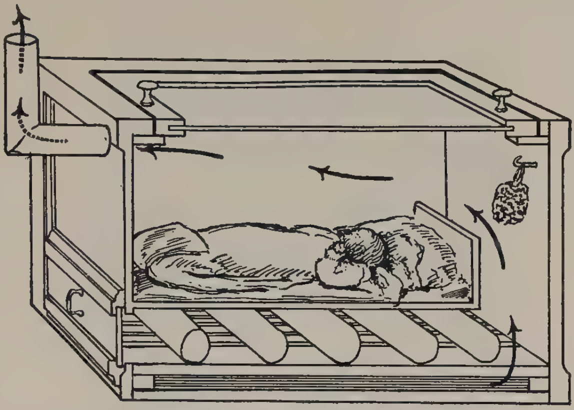

Fig. 138.—Tarnier incubator. First practical closed type incubator used in 1880.

A thermometer is fastened near the side window, so that it may be easily read, and a hygrometer is used to indicate the degree of moisture.

This type of incubator has in the past 10 years lost considerable of its early popularity, as is evidenced by a visit to most of the large hospitals. A great deal of this deserved unpopularity is due to the inability to properly ventilate them in the ward, and the necessity for furnishing a trained attendant. To properly supply these incubators with a free current of air, it is necessary to connect them so that they will receive a supply of air from outside of the building. To counteract the tendency of an insufficient air current in the absence of winds or when the wind is in the wrong direction, an electric fan should be incased on the outside of the building in such position that air may be blown directly through the incubator. This is difficult of arrangement when the incubators are located on the first floor of the building. When the station is located on the first floor, it is necessary to avoid the dampness and dust of the street level, and this can be accomplished by installing a large funnel 15 or 20 ft. above the ground level, some 24 to 36 in. in diameter to which is attached a 10-in. stack which can be connected with a cage at its base in which the electric fan is installed. From this point the air is blown through the system of incubators.

When such a considerable quantity of air is blown into the incubator system, it becomes necessary to filter it through several layers of cotton. This is best done at the side of the individual incubator.

The Cincinnati Hospital uses an electrically heated bassinette. The temperature is regulated by a series of electric lamps under the mattress. The apparatus consists of a double wall frame, within which heated air rises and escapes through small holes near the top. These openings can be opened or closed by a sliding damper as may be required.

In 1914 the writer designed an electric heated water-jacketed infant bed, Fig. 140, which combines the feature of the double wall water jacket, which has long been in use, with insulation to prevent external loss of heat and electric heating by a large plate with rheostat control.

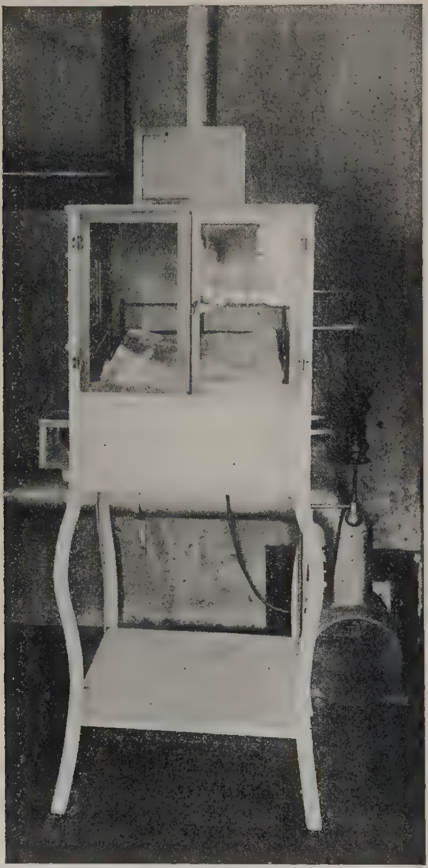

Fig. 139.—Lion type incubator (Couney model).

The fresh air is forced through a large air shaft by an electric fan on the outside of the building.

For hospital and home equipment the bed answers many requirements, because of its simplicity of operation in any well ventilated and moderately heated room. It can be used for the care of premature infants, for the protection of the newborn full-term infant immediately after delivery, and for infants suffering from hypothermia from other causes. The construction of the bed is such that it is intended for use in an ordinary ward or room, giving the infant the advantage of a most perfect room ventilation.

The following suggestions will aid in the practical application of this bed for use in hospitals or the home.

A special room should be provided. This has a practical advantage through the instruction of the nurses to consider this room as barriered, and will make a demand upon the nursing staff for observation of all of the rules of aseptic nursing.

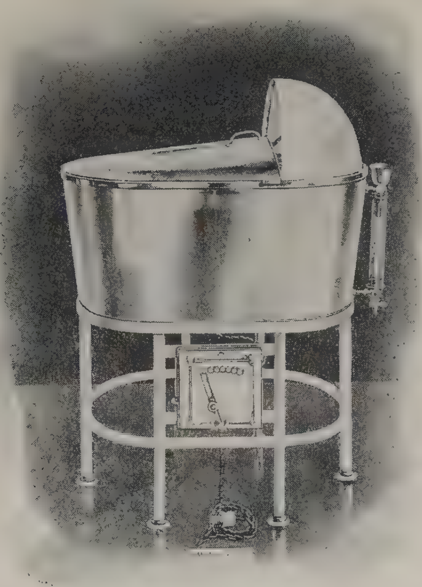

FIG. 140.—Electric heated, water jacketed infant bed. (Hess.) Bed complete with removable metal lid and collapsible hood in place. The hood and lid can be removed as indicated.

The room for premature babies should be supplied with an ample system of heating coils controlled by a thermostat for winter use, thereby facilitating the maintenance of a more or less stable temperature in the room which should, in so far as possible, range between 68 and 75°F.

The temperature within the room and bed should be read and charted at six-hour intervals, best at 6 a.m., 12 m., and 6 and 12 p. m. as the most likely times for maximum changes in the ward temperature.

Ventilation should be adequate but not excessive, and the room should be so constructed that the beds may be placed outside the line of direct air currents. This is accomplished by having the ventilating windows and transoms on one side of the room, while the opposite side is built with non-ventilating windows or blank walls.

Humidity in so far as the room is concerned will require little attention except at such time when considerable artificial heating is necessary. To supply the needed moisture during cold weather when ventilation of the room is more or less limited, a large evaporating pan should be in direct contact with the radiator coils. When these means fail to furnish the desired moisture, a wet sheet may be hung in the room and remoistened as indicated by the hygrometer.

In so far as possible the relative humidity should be kept at about 55 per cent. However, amounts less than this down to 40 per cent. will usually cause little or no discomfort or retardation of progress. It has been our experience that with a good free ventilation through open transoms or windows when the temperature of the room does not exceed 80°F., the normal water content of the air is quite sufficient and little or no attempt at influencing the room humidity is necessary. However, this will not answer the purpose where a closed room is used.

When a special room cannot be provided the bed should be kept in the nursery used for normal infants. They must never be brought into contact with infected infants because of the danger of crossed and mixed infections. On the other hand infected prematures should not be placed among well newborn infants.

All infants should be removed from this room to the nursery once daily, so that it may be thoroughly ventilated and cleaned by the use of soap and water. Before replacing the infants the air should be reheated to remove excessive humidity.

Home-made Heated Beds.—*Emergency Equipment.*—As many of the cases must be cared for in the home and in most instances without time or facilities to properly equip a nursery, every physician should have some definite ideas on the construction of a bed which will meet exigencies of the individual case. We have already spoken of the general care and equipment of a nursery unit in the home. A number of practical emergency beds have been described, the specifications of a few of which will be given more in detail.

A small wash basket well padded inside and outside by quilting into which is fitted a movable platform about 4 in. above the padded floor of the basket makes a fair emergency bed. Beneath the platform in the floor of the basket, hot water bottles or bags are placed which must be refilled from time to time. The removal of the bags for refilling which should be three or more in number and which are to be filled at different times, is facilitated by cutting an opening along the lower outer wall of the basket through which the water bags can be removed at will without disturbing the infant. A box can be built for this purpose to even better advantage.

Whether a box or basket be used, it must be provided with some form of cover for three-quarters of its upper surface. This may be accomplished by using a heavy blanket.

In such a bed the infant must be provided with proper clothes as previously described to prevent undue heat loss, and the bed should be kept in a well ventilated warm room, the temperature of which should range between 68 and 72°F. if possible.

Brown* describes the following practical home-made heated bed: Take a 24-in. wicker clothes basket and pad the bottom with non-absorbent cotton to a depth of 8 in. On top of this cotton fit a sheet of oilcloth, sewing the edges through the sides of the basket. On the oilcloth lay a double layer of white flannel and on the flannel a napkin of absorbent cotton. Six 12-oz. citrate of magnesia bottles with wire and rubber corks covered with flannel are filled with water at 110°F. and hung on the inside walls of this basket. A thermometer hung inside should register a temperature from 80 to 90°F. at all times. At night an oilcloth is spread over the foot half of the top of the basket.

Electric heating pads protected by copper jackets have been in use by the writer over a period of several years, and offer a valuable means of meeting emergency requirements. They are also valuable for use in the home where the temperature cannot be well regulated, after infants leave the hospital station. Electric heating pads have lost their popularity through the danger of fire from short circuit due to broken wires, and through the poor quality of the thermostat attachments of some of the pads. To avoid the danger of fire from short circuits in electric heating pads, a copper receptable is used, 16 n. long, 13 in. wide, and 1¼ in. high, into which a 12- by 15-in. heating pad is laid. To allow of a maximum radiation from the lid or upper surface of the same, the floor and sides are lined with asbestos sheeting, while the lid is not lined. The cord passes through a small rubber insulator at the side to prevent contact with the metal and injury to the cord. This simple device can be used temporarily in wards and homes where better facilities for the care of this class of infants are lacking. It is to be placed in the bottom of a basket or crib, under the mattress or pillow.

Litzenberg† has described a practical bed for home or hospital use for which the specifications are as follows: A box 24 in. long, 20 in. high, 18 in. wide. Eight inches from the bottom is a false bottom dividing the box into two chambers, the heating apparatus being in the smaller lower chamber and the baby in the upper one. The false bottom is the support for the bed of the baby and does not cover the whole bottom of the box, a space of 4 in. being left at one end for the circulation of hot air. The top of the box may be fixed on hinges or arranged to slide which is better. There is a pane of glass in the top so that the baby may be watched, and there are two ventilating holes near the end of the cover opposite the place where the hot air enters. An ordinary pillow is laid on the false bottom for the bed. The incubator is heated by bottles filled with very hot water and placed in the lower chamber through a small door in the side of the chamber. Fresh air enters this door, passes over the hot bottles, is heated and ascends by way of the 6-in. space at the end of the box to the baby's chamber and out through the ventilating holes in the top, giving a constant supply of warm fresh air. A thermometer is placed in the incubator beside the baby, or better beneath the first fold of the enveloping blanket.

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* Brown, Alan, Archives of Pediatrics, xxiv, No. 8, p. 609.

† Litzenberg, J. C., J. Minneapolis M. Ass. Mpls., 1908, xxviii, pp. 87–91.

By watching this thermometer a fairly constant temperature can be maintained by frequent filling of the bottles.

The room incubator or so-called giant incubator claiming to have all the advantages of little incubators without their inconveniences, was constructed in Lyon, France, for the first time in 1886 by H. Colrat. It consisted of a room 12 ft. long and 6 ft. wide. Its two main features were an attempt to hold a constant temperature, and a system of aeration permitting of renewing the air. It was, no doubt, a good innovation at that time.

In 1900, Arnaud of Turin introduced the hot-air room, and it found followers in other cities.

The incubator chambers built by Escherich and Pfaundler in Graz and Vienna, Brauer in Marburg, and Langstein in the Kaiserin Auguste-Victoria House in Berlin are all of the same type with added improvements. They are completely enclosed cells of glass and metal construction, having sufficient room for two or more infant beds, obtaining the air from outside and are provided with automatic regulation of gas heating, ventilation, and humidification. Between the cells and the nursery room there is a small space providing against cooling of the infant when the door of the incubator is opened. It is possible to change the clothing of the infants, to bathe them, and to feed them in the room.

Several clinics in the United States have built such rooms, among them Washington University of St. Louis and Michael Reese Hospital, Chicago.

In its primitive form an incubator room may be provided in a private home by heating the room to 75 to 80°F., at the same time making provision for moistening the air sufficiently by hanging wet cloths near the stoves or radiators. It is, of course, impossible to maintain a constant temperature and ventilation by such crude means, so that in conjunction with a more moderately heated room, 70 to 75, some type of individual bed for the further protection of the infant should be used.

The disadvantages of the larger incubator room have led in many clinics to their being discarded, among others that at Michael Reese Hospital, except in so far as they are used with a more simple form of heated beds. Some of the greater difficulties to be overcome are the automatic heat regulation; the cost of equipment and maintenance when only a small number of children are to be cared for, the distress caused the attendants when they are required to remain for a considerable period in the heated room, and most important the difficulty encountered in individualizing the care of premature infants of different ages and stages of development.

Specifications of Warm Room, Washington University, St. Louis.—The fresh air from outside is driven in by an electric fan. It then passes over a system of steam coils enclosed in a closed steel cabinet, and is moistened by steam escaping through a small valve within the cabinet. Thermostat contact is used. The air makes a complete circuit of the heated chamber and passes into a closed shaft and enters the room through small registers located in the shaft.

The used air leaves the room through the outgoing shafts of the ventilating system. The room itself is insulated and the windows double.

A thermometer and hygrometer are placed near a window and are visible from the corridor. A nurse records the room temperature and humidity on a chart every hour.

To hold the room temperature at approximately 80°F. and humidity at 55°F., regular inspection is necessary because of the unsatisfactory working of the thermostat.

DISEASES OF THE RESPIRATORY TRACT

Asphyxia Neonatorum.—Asphyxia is a condition produced by any interference with oxygenation of the blood. It may be present at birth or it may occur subsequent to that event. Asphyxia in the newborn is characterized by an absence or feebleness of respiration although cardiac action shows that life is present. Asphyxia occurring after birth is most frequently due to prematurity or to congenital weakness.

Etiology.—Asphyxia of the premature newborn may be due to any one of the many causes which interfere with the oxygen supply of the fetus either before or during labor.

In the extra-uterine variety of asphyxia the infant attempts respiration after birth but is unsuccessful. The reason for this failure may be due to the presence of mucus, blood or liquor amnii in the respiratory passages; to the presence of anomalies of the heart or lungs; to injuries of the skull; to the pressure from cerebral hemorrhage; or to inherent constitutional weakness associated with undeveloped respiratory muscles. In the premature infant the respiratory center is but insufficiently developed, the respiratory muscles are weak and the lungs are in a state bordering more or less closely upon fetal atelectasis. All of these factors favor the development of asphyxia, and the younger the fetal age of the infant at the time of birth, the more pronounced are these conditions, though it must be remembered that not all premature infants are debilitated (see "Atelectasis").

Cerebral pressure from injuries of the skull or from intracranial hemorrhage causes anemia of the medulla and consequently prevents stimulation of the respiratory center with resulting lack of respiratory activity, or with stimulation of the vagus with excessive slowing of the pulse, which interferes with the exchange of gases through the placenta or the lungs.

Symptoms.—The strong premature infant at birth behaves much as does the full-term healthy child; it breathes deeply, utters a more or less vigorous cry, and the skin which at first is of a purplish hue rapidly becomes pink. If asphyxia exists two sets of symptoms may present themselves, depending upon the variety of asphyxia, *asphyxia livida* or *asphyxia pallida.*

Further Course.—If an asphyxiated infant is revived it frequently remains somewhat apathetic, cries very little and does not nurse well, requiring artificial aid in obtaining nourishment. In the stronger infants, however, this condition tends to clear up, so that in a few days the cry is vigorous, the movements active and the ability to nurse is good. In the weakling, whether premature or full-term, such improvement is much slower. The poorly developed respiratory mechanism results in superficial and irregular breathing and the existence of areas of atelectasis tends to delay development of the lung. These weak infants may have breathed spontaneously at birth though not enough to have dilated the alveoli of the lungs to a sufficient degree and as a result repeated attacks of cyanosis occur. These attacks of cyanosis are accompanied by a condition of apnea which lasts a moment or longer, during which the infant ceases to breathe entirely. These attacks appear without warning and may be very frequent in the weaker infants during the first two weeks of life, and are evidently the result of lowered irritability of the respiratory center. The outlook for the infant in these spells is not good, despite the fact that treatment is undertaken, because they are an indication of inherent weakness in the individual. In those cases which are to recover these attacks of cyanosis become less and less severe and less frequent.

The after-life of these infants may be affected to some extent as the persistence of a degree of atelectasis renders them less resistant to infection.

Sequelæ.—Cerebral symptoms that develop later are not at all infrequent in children asphyxiated at birth and probably depend upon cerebral sclerosis secondary to minute intracranial hemorrhages. Developmental cerebral anomalies or injuries may, however, be primary causes of asphyxia and may later be evidenced by motor and psychic disturbances.

Diagnosis.—Asphyxia must be differentiated from hemorrhage of meningeal or cerebral origin occurring during prolonged or abnormal labor or after the application of forceps. The symptoms of a slight hemorrhage resemble those of asphyxia, the breathing being very superficial with frequent lapses into stupor. Convulsions occasionally occur and the pulse may be slow or fast. Continued slow pulse with the occurrence of coma and convulsions speak strongly for a cerebral hemorrhage, especially after a prolonged labor or the application of forceps. The differentiation is extremely difficult during the first days of life in premature and weak infants, and death frequently results before the etiological factor is ascertained. Delmas* recommends lumbar puncture as a diagnostic and therapeutic measure. The results obtained have been unsatisfactory.

Prognosis.—The outlook for strong prematures suffering from asphyxia livida is good, the majority recovering under proper treatment. In the weaklings it is always grave. In asphyxia pallida the prognosis is bad, the infant invariably succumbing if left to itself. If the heart action improves while attempts at resuscitation are being made, it is a favorable sign. Endeavors to revive the infant should be kept up until the heart ceases to beat. At all times undue violence must be avoided, all attempts at resuscitation being applied gently and at regular intervals to avoid visceral injury. If cerebral hemorrhage is combined with asphyxia the outlook is very poor.

The cause of death in asphyxia may be a recurrence of the asphyxial attacks, lowered irritability of the respiratory center, atelectasis of the lung or blocking of the air passages by inspired foreign matter or cardiac failure.

Treatment.—The treatment of asphyxia is concerned with clearing the respiratory passages and supplying oxygen to the tissues. In the milder cases the finger is gently introduced into the pharynx, or the throat stripped downward, while the child is held in an inverted position, sufficient to clear out the obstruction to respiration. In the cases of asphyxia livida there is usually mucus in the trachea or bronchi, and this can frequently be removed sufficiently to allow of respiratory activity by inverting the infant and introducing a catheter as far as the upper opening of the larynx. Only in the larger infants is it possible to pass the catheter into the larynx. Suction is made with the lips and the mucus is drawn into the catheter. The danger of contracting syphilis must be remembered. Occasionally, it is necessary to repeat this maneuver several times.

Once the passages are cleared of mucus, the reflex stimulation of respiration by external irritation is attempted. In the milder cases the back and buttocks of the suspended infant are gently slapped, cold water is sprinkled over the body, or the baby is rubbed with a warm cloth. In the severer cases the infant is immersed in hot water at a temperature of 40.5°C. (105°F.) for a few minutes and then in a cool (90°F.) bath for an instant. The warm bath relieves the vasoconstrictor spasm and the overloaded heart, the blood being brought to the surface. Weak mustard baths; warm enemata and careful compression of the chest are all advocated.

In the severest cases cutaneous stimulation is not sufficient and it becomes necessary to resort to artificial respiration. This must be done slowly, regularly and gently.

Cyanosis.—Of all functions of the premature infant, that of respiration is usually the least developed at birth, evidencing to a marked degree the general lack of development of the central nervous system. Failure on the part of the respiratory apparatus to respond in a sufficient manner to the needs of the infant is the most frequent cause of symptoms of the gravest nature in these weaklings and indeed not seldom of death itself.

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* Delmas, Jean, Le Progrès medical, xl, S., 1912, pp. 88–89.

The underlying factors in the production of cyanosis may be divided into inherent and extraneous. The *inherent* causes are:

1. Lack of development of the central nervous system, especially of the respiratory center.
2. Weakness of the general musculature and softness of the ribs.
3. Persistence of fetal atelectasis which tends to delay development of the lungs.
4. Congenital malformations of the heart or great vessels or myocardial asthenia.
5. Malformations of the respiratory tract or of the diaphragm.
6. Diseases or compression of the air passages.
7. Injuries of the skull or cerebral hemorrhage.
8. Obstruction to nasal breathing.
9. A birth-weight below 1200 gm. These infants almost invariably suffer from attacks of cyanosis.
10. Cooling of the body is given as a cause by Budin, but many infants have a temperature of 95°F. without the occurrence of cyanosis.
11. Elevation of the body temperature to more than 102°F. is given by Zahorsky as a cause.

In the premature infant the causes among the above which are chiefly operative in the production of the characteristic attacks of cyanosis are the weak respiratory muscles, the softness of the ribs, the under-development of the centers of respiration and the presence of fetal atelectasis.

Involvement of the heart is ordinarily of secondary occurrence, the diminished amount of oxygen in the blood resulting in a slowing and weakening of the heart's action. The atelectasis which is so frequently present, tends to hinder the closure of the foramen ovale and the ductus Botalli and these defects in turn predispose to cyanosis.

The *extraneous* causes include:

1. The aspiration of food or vomitus into the larynx or trachea. The lack of development of the pharyngeal and laryngeal reflexes are responsible for the food reaching the air passages, and the lack of reflex cough prevents its being ejected. Pneumonia not infrequently follows the aspiration of such foreign particles.
2. Distention of the stomach from overfeeding. This is one of the most common causes of cyanosis and death in premature infants. This leads to interference with the action of the diaphragm (Birk).
3. Meteorism, due to gastric and intestinal stasis.
4. Attempts at drinking are often followed by cyanosis, either the direct result of the mechanical prevention of respiration or secondarily through the lessened oxygen content of the blood resulting in a lack of stimulation of the respiratory centers (von Reuss).
5. Under-nourishment is strongly advanced by Budin as causative of cyanosis, and he has shown that with increased feeding these attacks

stop. Without doubt improvement in the general state of nutrition improves the working of both the respiratory centers and the muscles of respiration, but that increased feeding is of value in overcoming this complication, except indirectly, is hard to believe.

6. An insufficient supply of water.
7. The occurrence of a local or general infection.

Symptoms.—Often times, without apparent cause, attacks of cyanosis appear with frequency during the first few weeks of the life of the premature or weakly infant. Usually without warning the respirations, which have previously been superficial and irregular, become still weaker and then cease entirely for a minute or longer, somewhat resembling the Cheyne-Stokes type of breathing. Accompanying the apnea is a deep cyanosis which gradually disappears as breathing is resumed. Not infrequently, if immediate steps to restore the respiratory activity to something like the normal are not taken, the infant dies; in other cases breathing is spontaneously resumed and the attack passes off, leaving the infant more or less prostrated. Care must be taken not to pronounce it dead before examining for heart sounds. In a few hours or days cyanosis recurs, the attacks gradually increasing in length and severity despite treatment, until death occurs; or they become less frequent until they cease entirely.

Occasionally the attacks are preceded or accompanied by convulsions. Generalized edema sometimes develops.

Diagnosis.—From congenital cyanosis due to other causes, or acute affections of the respiratory tract with cyanosis, these attacks are differentiated by the history or other evidence of premature birth, and the frequently accompanying cyanotic edema, the respiratory weakness, absence of the normal vesicular murmur, particularly over the bases and the tendency to a subnormal temperature.

Prognosis.—The prognosis of cyanosis in the premature infant varies directly with the severity of the attacks which in turn are more or less directly dependent upon the fetal age and physiological development, the ability of the infant to maintain its body temperature, the quality of the food, and the ease with which the infant digests it.

In no other condition to which these infants are subject is the previous training and experience of the attending nurse in the care and handling of this class of cases, of such vast importance.

Treatment.—A premature infant must be carefully watched for signs of cyanosis, otherwise it may be found dead in bed. Should an attack occur while the child is being fed, the proceeding must be stopped and efforts made to restore respiration. The first step is to ascertain if there is any obstruction in the upper respiratory passages. Should inspired food or vomitus be present, an effort must be made to dislodge these particles. Inserting the little finger into the pharynx while the child is in an inverted position, often serves to clear out the respiratory tube, and then slight cutaneous stimulation by pinching, friction, or gentle slapping is often enough to re-initiate breathing.

Again, exhaustion of the infant may be solely responsible for the cyanosis. In these cases artificial respiration should be tried; the chest being rhythmically pressed upon, or one of the other methods of artificial respiration may be tried. Simple compression of the chest may be tried without removal from the incubator or bed, though placing the infant on a table will be found more serviceable. *Great gentleness is imperative.*

The use of oxygen is of value in quickly reducing the degree of asphyxia after breathing is once established, although it will not of itself restore that function. A tank should be kept by the infant's bed and any sign of approaching asphyxia should be the indication for a generous shower of oxygen. The continued use of the gas when properly applied is advocated as a valuable measure in the checking of attacks. About 80 to 100 bubbles of oxygen gas from a partially protected mask should escape in close proximity to the infant's mouth.

Aromatic spirits of ammonia in one-half to two drop doses, diluted, is of value, and nitroglycerin, one drop of a 1:1000 solution may be placed on the tongue. The use of camphor, caffein, atropin or other respiratory stimulants hypodermically does not offer much practical help.

Sprinkling the baby with cool water will occasionally stimulate respiration and as this means is always at hand, it should be kept in mind.

Infants suffering from repeated attacks of cyanosis should be immersed in a hot bath at a temperature of 102 to 105°F., and subjected to gentle friction, more especially along the spinal column. The infant may be kept in the bath for from a few seconds to several minutes, when it should again be placed in its warmed bed, avoiding all chilling. The efficiency of the bath may be increased by the addition of a teaspoonful of mustard to the gallon of water. Care should be taken to prevent aspiration of the bath water, or its entrance into the eyes, and the danger of infection of the umbilical cord, although not great must be borne in mind. The bath may be repeated as indicated.

In our own experience the warm mustard bath has proven one of the most satisfactory means of overcoming prolonged attacks. It is quite evident that the facilities for preparing the bath must be prearranged and great care taken to keep it at an even temperature throughout the immersion. To facilitate handling and to prevent undue manipulation during the cyanotic attacks, the infant should be wrapped in a blanket.

It cannot be too strongly emphasized that the manipulations used to relieve the cyanosis should be the minimum necessary to accomplish the result as cyanotic infants react poorly to trauma. After an attack is over the infant should be placed for a short time in a hot bath in order to overcome the tendency to a reduction of temperature by the previous manipulations. Afterwards it is also necessary to supervise carefully the feeding in order that two things may be accomplished:

First, that the occurrence of further attacks of cyanosis due to mechanical obstruction by food may be prevented; and secondly, that the nutrition of these weaklings may be immediately bettered and thus the cyanosis indirectly controlled.

The prevention of cyanosis may be aided in several ways. The too rapid taking of food or distention of the stomach by overfeeding must be avoided (see "Feeding"). Underfeeding in cases where too frequent feeding is undesirable can be avoided by catheter feeding at longer intervals, although the maximum food quantities must be carefully ascertained by starting with minimum feedings, carefully increased according to the infant's tolerance. Catheter feeding is not well borne by all infants and may occasionally in itself induce cyanosis. The strength of the infant should be built up as rapidly as possible and the temperature of the body should be maintained by the use of the heated bed inasmuch as a lowering of the body temperature not only favors the development of cyanotic attacks, but makes them more severe when they do occur. The use of oxygen may be of value.

Insufficient supply of fluids should be avoided by the administration of water where the fluid intake is less than one-sixth of the body-weight during the 24 hours.

Meteorism may be relieved by small quantities of low saline enemata, part of which may be left in the rectum to good advantage where the fluid intake per mouth is insufficient to meet the body requirements.

Gastric lavage must occasionally be resorted to as a means of last resort in over-distention of the stomach with paresis of its walls and should be performed with the infant's head at a lower level than the body to prevent aspiration of stomach contents, as passage of the tube very frequently results in vomiting. This procedure is always associated with great danger during a cyanotic attack. Occasionally the gas can be relieved by simple passage of the catheter into the stomach with slight pressure from without over the epigastric region.

Atelectasis.—Atelectasis is also spoken of as acquired asphyxia though it may be congenital as it is a persistence of the fetal state in all or in part of the lung. In the congenital variety the lung is not entirely expanded at birth, while in the acquired form collapse of the previously expanded lung occurs. The congenital variety is seen chiefly in the premature and debilitated, the respiratory muscles not possessing sufficient strength to inflate the lungs. The acquired form is due to obstruction of a bronchus, or of alveoli, to a deformed spinal column, or to a pleural exudate.

Atelectasis is to a degree physiological during the first few days after birth, gradually disappearing with increasing strength. Days elapse before the volume of the lung is increased to the point at which the organ is fully expanded, while in the premature and weakly this expansion takes still longer.

Atelectasis is usually associated with asphyxia at birth, but is overcome often entirely by the means used to revive the infant. In the weak, the methods used are not enough to cause complete expansion of the lung and collapsed areas persist, the soft and yielding thoracic wall and poorly developed respiratory muscles of the premature both favoring the non-expansion.

Pulmonary atelectasis also occurs after cerebral hemorrhage, due to injury to the respiratory center, and is characterized by small respiratory excursions and slight exchange of gases. In the premature the irritability of the respiratory center is low *a priori*, while in those suffering from natal asphyxia it is lowered by the asphyxia (von Reuss*).

Differential Diagnosis.—A number of conditions must be considered in the differentiation of atelectasis, the more important of which are the following:

General debility with quantitative and qualitative lack of development attended with impaired respiratory, cardiac and digestive functions. This is commonly associated with lack of development of the thoracic wall and a tendency to collapse on the part of the costal cartilages, and a poorly developed respiratory musculature.

Cerebral injury associated with hemorrhage is one of the most difficult pathological conditions to differentiate, because of the tendency toward involvement of the respiratory centers, more especially in basilar hemorrhages. A careful inquiry should be made for a history of opisthotonus and clonic contractions of the extremities or facial muscles.

Hyperplasia of the thymus and occasionally the thyroid gland, with associated stridulous respiration, retraction of the diaphragm and local physical findings. When the chin is brought down upon the chest respiration becomes more difficult, and in turn is made easier if the head is bent back.

Aspiration of foreign matter or food with lack of expulsatory effort resulting in cyanosis may lead to error in diagnosis.

Underfeeding, with secondary asphyxia.

Congenital diaphragmatic hernia.

Prognosis.—This depends upon the degree of atelectasis which in turn usually depends upon the degree of debility of the child. When accompanied by attacks of asphyxia and cyanosis which appear with frequency during the first two weeks of life, the outlook is bad, despite the institution of proper treatment, as these attacks commonly result fatally. In favorable cases they become less frequent and finally cease. Pneumonia in atelectatic areas often leads to a fatal issue.

Infants who have suffered from congenital atelectasis may remain in delicate health for a long time, although many ultimately recover completely.

Treatment.—The physical condition of these weaklings is oftentimes so precarious that undue roughness in the application of restorative measures can work infinitely more harm than they may do good, and so it must be remembered that the less the manipulation necessary to overcome the cyanotic attacks, the less is the danger of injuring the infant at this critical time either by overstimulation mechanically, or by medication. The object of treatment is directed toward the expansion of the lungs through deep breathing. This is done by crying, and if the child does not cry strongly every day, it should be made to do so.

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* Von Reuss, "Diseases of the Newborn," Julius Springer, Berlin, 1914.

The infant should not be allowed to lie quietly in one position, but its position must be changed frequently and the child picked up several times a day. Particularly where many infants are housed with but little individual attention atelectasis is seen most frequently. The further treatment should be similar to that advised for attacks of cyanosis.

CONGESTION AND INFLAMMATORY CHANGES IN THE LUNGS

Congestion of the Lungs.—Congestion of the posterior lower portions of the lung most commonly results from long continued rest without change of position, congenital or other anomalies of circulation. At first there may be extravasations of serum or blood in the alveoli and later, especially in the greater degrees of congestion, tissue infiltration.

Clinically the condition is manifested by disturbances of respiration, shallow breathing, asphyxial attacks, and even impairment of resonance which may be confounded with inflammations or atelectasis (Hayashi*).

Congenital Pneumonia.—That congenital pneumonia may exist seems to be well substantiated, although the number of cases reported in which the infection was hematogenous and transmitted by way of the placenta is small.

Post-natal Pneumonia. *Etiology.*—Bronchitis during the first few days of life may be the result of aspiration of infectious material, or it may accompany a general septic infection. The fact that the vaginal secretion of the mother always contains microörganisms offers every opportunity for infection, should aspiration occur during the infant's passage through the birth canal. Infection of the bronchi may reach the child from an infected mother or attendant, or from a third person through the agency of feeding utensils, spoons, or other articles. Infections in the upper air passages may spread by direct extension to the deeper structures of the respiratory passages and there occasion a bronchitis or a bronchopneumonia.

The two factors which favor the occurrence of pneumonic inflammation are the existence of atelectatic areas, so common in the lungs of the premature or weakling, and the frequency of aspiration of food or vomitus in the debilitated. These weaklings are often unable to nurse from the breast or to take milk from the bottle, even when the milk is fed to them with the spoon or medicine dropper. Because of the imperfectly developed swallowing and pharyngeal reflexes, some of the milk runs into the trachea and then is aspirated into the bronchi, carrying with it bacteria from the mouth and throat. The richness of the atelectatic portions of lungs in blood and tissue fluids make them a most favorable medium for the multiplication of the invading bacteria.

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* Hayashi, A., Mon. f. Kinderheilk., xl, 1912, p. 224.

Pneumonia in atelectatic lungs, aspiration bronchitis and bronchopneumonia are often the immediate cause of death in the premature and congenitally weak, being very frequently terminal infections.

Symptoms.—The onset is most often insidious in the weak newborn. At first there is noticed possibly a slight nasal discharge and a cough of varying severity. Soon increased frequency of respiration makes its appearance accompanied by dilatation of the alæ nasi. The cough in the more mature becomes worse and the respiration increases to 60 or 80 per minute. Now and again slight attacks of cyanosis occur, which in the severe cases are correspondingly more marked. There is great restlessness in older infants with inability to sleep, and the cyanosis becomes continuous. Convulsions occur with more or less frequency, while the temperature may be slightly elevated or may be subnormal even in the severest cases. There is a marked loss of weight, the stools become dyspeptic, and greenish with mucus and undigested particles. The prostration may be extreme.

There is often a singular lack of symptoms and the disease may go unrecognized. The respirations range from 40 to 60 or 80 to 100 per minute, but are usually not labored, the pulse rate is increased to 140 or 160 and sometimes cannot be counted; the cough may be absent entirely, and there is often apathy and even deep stupor. The course in these infants is usually acute, either immediate improvement or death occurring.

The severity of these early symptoms is to be explained either on the basis of the sudden intense congestion of the small alveoli interfering with the bronchopulmonary apparatus almost as much as does consolidation, or on the intensity of the infection.

Diagnosis.—The diagnosis is often very difficult in infants. The physical symptoms of bronchitis and consolidation are frequently absent or quite anomalous in character. It may be necessary to make the infant cry or breathe deeply in order to bring out the various abnormal sounds. The presence of râles or evidence of congestion or consolidation as shown by impaired resonance and attacks of cyanosis, make a diagnosis of pneumonia; but it will usually be found postmortem that the morbid processes are much more extensive than one is warranted in assuming from the physical findings.

Pneumonia may, in these infants, be easily confounded with atelectasis. If the premature infant is strong and possesses a loud cry, congenital atelectasis may be excluded; in the weak the latter condition is most commonly present and the physical signs of pneumonia are absent. It must be remembered that the two conditions may exist side by side in the same infant.

Prognosis.—This is usually unfavorable in the first days of life particularly when of sudden onset. If the inflammation complicates infection in the upper air passages, such as a rhinitis or bronchitis, the outlook is better. Infection with the staphylo- and streptococci offers a more serious prognosis than with the pneumococcus. Involvement of a large portion of both lungs or an extremely weakened condition of the infant, both militate strongly against recovery. The younger the infant the higher the mortality, and when hypothermia exists death usually occurs soon. Cases which run their course with little or no temperature are usually fatal, probably because they occur in infants who are very feeble, of low vitality, with limited resistance to infections.

Treatment.—Prophylaxis.—The prevention of pneumonia in the premature requires that the little weaklings shall be protected against infection from every source. The lungs are most frequently the site of bacterial invasion, as there the organism finds a most favorable medium for its growth. In the adult there exists at the entrance to the respiratory tract defensive agents capable of stopping the invading bacteria but in the premature these defences are absolutely rudimentary and consequently offer but slight impediment to the entrance of pathogenic germs (Delestre*).

The transmission of respiratory infections occurs by means of infected hands or other objects or through the medium of air. No one suffering from any infection of the nasal or respiratory passages should handle the infant. If the mother is affected with a coryza or bronchitis she should take care that her hands are not contaminated with nasal or bronchial secretions and that she does not breathe, or cough in the infant's face. In institutions where many babies are taken care of by one nurse, the hands of the attendant should be washed before the handling of each baby. Isolation of the premature should be practiced if respiratory affections exist among the other members of the family, or in the common wards if the infant is in an institution. Only if the attendants are thoroughly trained in the principles of aseptic nursing is it safe to leave the infant in close proximity to others suffering from respiratory affections. These of all infections are hardest to prevent. All utensils should be individual and should be sterilized before use; feeders, spoons, glasses, nipples, bottles, stomach tubes, etc., must all be boiled before they touch the child or its food. As soon as these babies can be removed from the incubators they are kept in large, well ventilated rooms, which are not overheated. They should be given the benefit of open air and sunshine as their development warrants. In favor of the open-air treatment is the fact that most of the late deaths occur during bad weather.

General Treatment.—The treatment of pneumonia is preëminently that of watchful expectancy, and over-treatment must be avoided, as these feeble infants are unable to withstand over-manipulation or stimulation. As a rule pneumonia in robust infants is an acute self-limited disease, but in premature infants the course is apt to be somewhat subacute without the tendency to limitation. The indications in the treatment are to support the heart and conserve the strength. The feeding problem is difficult at any time in the premature and during an attack of pneumonia it becomes doubly difficult.

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* Delestre, Etude sur les infect. des prématures, Thèse de Paris, 1901.

The hygiene of pneumonia requires that the child receive plenty of fresh air, and to insure this in an incubator of the closed type is difficult. The use of the open type in part overcomes this difficulty. The position of the child should be changed frequently in order to obviate any tendency to hypostasis. If the sick infant has been housed in a closed incubator with questionable ventilation it should be removed to an open, well-heated room, and placed in a properly warmed crib or incubator bed. The prime indication is for the promotion of elimination and sufficient administration of inert fluid. Stimulation of the respiratory tract is best accomplished by mild counterirritation to the chest and the use of hot applications to the extremities. The use of drugs such as cardiac and respiratory stimulants is not to be regarded with favor but strychnin sulphate in $\frac{1}{500}$ gr. (0.00012 gm.) doses, or atropin sulphate in $\frac{1}{1000}$ to $\frac{1}{3000}$ gr. (0.00006 to 0.00002 gm.) doses given hypodermically may be of some help. The use of whisky or brandy is permissible in quantities varying from 3 to 10 drops every two or three hours depending upon the indications. Aromatic spirits of ammonia in one to five drop doses is one of the best stimulants at our command. Both the whisky and the ammonia should be given well diluted in at least eight parts of water. In case of emergency, of sudden heart failure or of weakness accompanying a sudden fall in temperature, the use of camphor-in-oil 2 to 10 minims to the dose given hypodermically will be found to be a rapidly acting, reliable heart and respiratory stimulant.

If the infant shows a marked rise in temperature, the use of hydrotherapy may be considered. Temperatures up to 103 degrees are well borne, and do not require interference. As a general rule the temperature tends to remain subnormal in these weaklings and cool or even tepid baths must be avoided and instead warm or hot mustard baths resorted to. Even if there is an excessive amount of fever, should it be accompanied by a cold surface, feeble pulse, and shallow respirations, cold is contra-indicated. The hydrotherapeutic measure used for the reduction of an unduly high temperature is the tepid pack. The use of cold baths or packs is probably never justified in the premature or weak infant. The temperature of the tepid bath may range from 100 to 105°F. depending upon the condition of the child.

The treatment of *attacks of collapse with cyanosis*, which are so frequent in the atelectatic prematures, should be prompt. The infant should be immediately placed in a mustard bath (one teaspoonful of powdered mustard mixed with 1 gal. of tepid water being of sufficient strength), of about 102 to 106°F. together with gentle massage. Respiratory and cardiac stimulants may be needed. Oxygen should be administered continuously.

Disturbance of the nervous system, occasionally so prominent in older and stronger children; is not marked in the premature during a pneumonic process. When present mild hydrotherapy offers the best results.

The use of the coal tar products is contra-indicated.

The diet is an extremely important part of the treatment of pneumonia and will be considered under "The Feeding of the Premature."

Frequent changes at regular intervals of the infant's position in its bed are imperative to successful care of pneumonia in the premature.

DISEASES OF THE GASTRO-INTESTINAL TRACT

In a consideration of this very important chapter as relating to the premature infant, we must first recognize the possibility of congenital malformations and other prenatal factors which might have an important bearing on the function of the digestive organs; secondly, the lack of proper physiological development necessarily present in the prematurely born, the importance of which varies inversely with the fetal age; thirdly, post-natal pathological conditions, developing in the gastro-intestinal tract; and fourthly, the importance of systemic infections in their influence on the processes of metabolism.

The use of the term "nutritional disturbances" rather than that of "digestive disturbances" is beyond any question more generally applicable to the premature than to any other stage of life, as in these individuals the rapid development of general nutritional disorders is the rule following even moderate causes.

It cannot be too strongly emphasized that the immediate institution of the proper hygiene, and the establishment of the proper prophylaxis toward the prevention of nutritional disturbance by the early administration of human milk whenever possible, are absolutely necessary to avoid disaster. It cannot be disputed that a great number of premature infants die, not because their organs lack that degree of maturity necessary to proper functions, but because of early neglect, either through lack of adequate facilities or ignorance of exact methods of feeding and care.

Functional Insufficiencies of the Gastro-intestinal Tract Wholly or in Part Dependent on Lack of Development

Difficult Nursing.—The causes of difficult nursing are to be found either on the part of the infant or on the part of the mother, or both.

The Infant.—Various factors may enter which may make nursing difficult or even impossible. Some of these will be treated under the chapter on "Methods of Feeding" (page 468). Of the malformations, those offering the greatest difficulty are cleft palate, harelip, and nasal deformities due to lack of cartilaginous development. The tendency to sleep constantly is often very perplexing. General weakness and lack of muscular development in the poorly developed premature are not infrequently sufficient to make nursing impossible. Infections of the mouth resulting in thrush, stomatitis, and ulcerative processes are always of serious import. All conditions interfering with proper respiratory functions, whether due to lack of development, such as atelectasis or pulmonary infections interfere with the proper taking of food. These are but a few of the many complications which may be cited as impeding proper nursing.

The Mother.—On the part of the mother the various pathological conditions of the nipples and breast must be given due consideration.

Anorexia.—Premature infants born in the seventh and eighth months rarely show a disposition to feed spontaneously during the first days of life, and in a large proportion of cases we are forced to administer the food without the infant taking an active part. Only a small proportion of the infants weighing between 1000 and 1500 gm. are able to nurse without assistance, and very few of them, unassisted, are able to suck with sufficient strength to take food from either the breast or the bottle. A very interesting fact which we have noted in premature infants weighing under 1500 gm., and occasionally in even larger infants, is a tendency to attempt at least to nurse spontaneously during the first two or three days of life, during which they, however, receive very little food. This period is followed usually about the third day by a marked somnolence, during which they show little or no inclination to nurse. This is usually associated with a rather rapid loss in body-weight due to underfeeding. At later periods not infrequently infants show a repugnance toward food, which may follow periods of overfeeding or be seen in the course of the gastro-intestinal or systemic infections. At whatever stage of the infant's development anorexia is seen it must be given the gravest consideration and every attempt made to administer food sufficient to meet the demands of the organism. Gavage must be resorted to if the less drastic methods of feeding are unavailing. It has been our experience that occasionally the omission of one or two feedings with the administration of a one-half strength physiological salt solution per mouth will result in the further stimulation of the appetite probably by producing thirst.

During this period the fluids should be given in sufficient quantity to meet the infant's needs, approximating one-sixth to one-fifth of the body-weight daily when possible, inclusive of the water. The addition of one to three drops of brandy is often a beneficial stimulant.

Inanition Fever.—Unquestionably hyperpyrexia as seen in the first few days of life and during the time when these infants are receiving a minimum of food need not necessarily be due to inanition. Many are undoubtedly due to infection or toxic products which enter the circulation through the gastro-intestinal tract. The products of decomposition as seen during the period of change from the meconial flora to the milk flora can undoubtedly give rise to hyperpyrexia, as is also true of the toxic products formed by decomposition of milk.

The effect of products absorbed from the intestinal tract on the parenteral cells, as well as the by-products due to the rapid changes seen in the body tissues, may, any and all of them, following their absorption, give rise to increase in body temperature. Occasionally, one sees cases of hyperpyrexia in the premature and in the newborn in whom the high temperature cannot be due to the surrounding artificial heat, and who make a rapid recovery without after-effect by simply increasing the fluid intake, of milk or water. The most striking cases that are seen are those in which water insufficient to meet the body needs has been given.

Treatment.—This consists in the administration of fluids equal to at least one-sixth of the body-weight of the infant in 24 hours and the administration of food as per the rules on "Feeding of Premature Infants during the First Ten Days of Life." Of equal importance is the prevention of overheating by excessive external heat. When doubt arises as to the causative factor in so-called "Inanition Fever" a small dose of castor oil (5 to 10 minims) together with a colonic flushing with a saline solution should be given in addition to the increase in the amount of water and human milk administered.

Vomiting.—In general the vomiting in the premature is of greater or lesser importance depending upon its intensity, and the result upon the general state of nutrition. Vomiting must be considered only as a symptom and not primarily as a disease, and again as a symptom which in its development is influenced by many factors peculiar to the premature infant. The relatively vertical position of the stomach in the sixth, seventh and eighth months as described under the "Physiology of the Premature Infant," is a factor of considerable importance, as is also the poorly developed sphincter at the cardia.

Of equal importance is the fact that most of these infants are fed mechanically in amounts theoretically correct for their weights and ages. But these same quantities may not agree with nature's idea of sufficiency, thereby leading to a rapid overfilling of the stomach through catheter and other mechanical means of feeding. Again the tendency toward abdominal distention and the frequent handling and manipulation of the infant all tend to promote regurgitation.

Vomiting persisting beyond the first or second week, even when varying in frequency and intensity, is likely to result in a considerable degree of under-nourishment. There is also the added danger in the case of premature infants with a minimal development of reflex irritability, that due to the lack of proper response on the part of the laryngeal reflexes, the regurgitated food may be aspirated with resulting sequelæ, such as cyanotic spells, asphyxia or even pulmonary infection.

Because of the great danger of underfeeding in the presence of the small food intake there is the gravest danger of weight losses, with the consequent development of inanition.

Etiology.—Previous to the taking of food the infant may vomit the various fluids such as liquor amnii and blood, which may have been swallowed during labor. These may easily be recognized by their character. Following the intake of fluid many factors must be considered, such as atresias in the digestive tract, excessive feedings, which may be due to too free nursing from an easily secreting breast in the older premature infants, or too rapid feeding of large quantities mechanically given to the smaller infants.

The dangers of compression of the abdomen due to improper holding or excessive handling of the infant are usually overcome by feeding in the bed.

The toxic vomiting as seen in the infants of eclamptic mothers and in the presence of sepsis, as well as hematemesis, will receive further consideration in the discussions on these topics. Vomiting may at any time become of serious moment and should always be given proper consideration. The relative loss of food as foretold by the scale, by weighing before and after feeding, and after vomiting, the weight curve and careful observation of the stools will give the best indications for therapeutic interference.

Treatment.—In the majority of cases there is no indication for active treatment. The occasional or even more or less regular "spitting" in the presence of a normal gain in weight and general well-being need receive little or no attention. However, when vomiting is persistent and is attended by stationary weight, which is equivalent to loss in weight in older individuals, or when it is associated with nausea or is explosive in character or contains bile, blood and other matter foreign to the normal stomach content, it should receive prompt and careful attention. No set rules for treatment can be prescribed, as the etiological factors in each and every case must be considered individually. The following general principles of treatment will in a great number of cases prove sufficient:

1. The infant should be subjected to a minimum of handling. It should, whenever possible, be fed without being removed from its bed, or where handling is necessary, all violence should be avoided.

2. The recumbent position with the head and shoulders slightly elevated assists in overcoming the tendency to regurgitation in the presence of a weak sphincter at the cardia.

3. Regulation of feedings is primarily indicated. This should cover first the number of feedings and the interval between feedings, and the amount of the individual meal. Not infrequently an infant who is receiving quantities too great for the stomach capacity will cease vomiting upon simple reduction of the size of the individual meal. Again it may be necessary to decrease the number of feedings, thereby lengthening the intervals between feedings. Furthermore, large meals at long intervals may be replaced by small meals at short intervals with a very beneficial result. Where the infant is nursing at the breast, simply reducing the time allowed for nursing in many instances will accomplish the desired end. The same may be true as regards the slower administration of the individual feedings in the bottle-fed.

4. In infants nursing directly at the breast where the shortening of the period of nursing is insufficient to control the vomiting, drawing the milk by expression or by breast pump and feeding a measured quantity which can be retained, either by hand or catheter, is often successful.

It is customary to start such feedings by giving 2 to 10 gm. at short intervals, 12 to 18 in 24 hours, preferably, although not necessarily, of freshly drawn milk, following this by gradually increasing quantities, and as soon as the proper quantity for growth is retained, lengthening the intervals, returning to direct nursing when the infant's general condition allows of the same. In the more severe cases the human milk may be boiled or a skimmed human milk should be used.

Under these conditions the milk supply should be protected through the emptying of the breasts by expression to prevent their drying up.

The question of even temporary starvation in the premature infant is one of serious import and should only be practiced after the most careful consideration, because of the rapidly developing apathy in this class of infants. The presence of so-called "hunger-stools" consisting of a brownish, stringy mucus with little or no food residue, is as a danger signal of almost equal importance with loss in weight.

If the above suggestions fail to accomplish the desired end, rather than to institute a starvation diet we prefer to empty the stomach by careful lavage, using a weak sodium bicarbonate or saline solution, and before withdrawing the catheter placing a small feeding of human milk into the stomach. Lavage is practiced not so much with the idea of removing any decomposed food content, but because of the sedative action on the mucosa. The dangers of gastric irritation from repeated introduction of the catheter in careless hands must, however, always be remembered, also the dangers of promoting cyanotic spells, through the careless deposit of fluids in the pharynx and larynx. In the infants artificially fed the problem is far more serious, and offers for its solution greater difficulties unless human milk can be obtained. In our own experience a well-boiled milk, in which the casein has been precipitated as a fine flocculent curd by the addition of rennet, has given the best results when human milk was not to be obtained (see "Preparation of Chymogen Milk"). Diluting the milk thus prepared before feeding, or skimming before boiling may also be of benefit.

Diarrhea.—Constipation is the exception; loose stools or a tendency toward diarrhea in both the breast-fed and the artificially fed premature infants is the rule. Therefore, frequency of bowel movements, especially in the breast-fed may be entirely physiological and unassociated with fever, vomiting and other evidences of gastro-intestinal disturbances. However, they may be due to contaminated food or intestinal infection, and every case should, therefore, be carefully studied so that evidences of deep lesions may be immediately observed. In the infant nursed by its mother the colostrum will almost invariably result in frequent bowel movements. This is one of the prime reasons in the selection of wet nurses, who have passed at least two or more weeks of their puerperium. The early milk also has a tendency to be high in its carbohydrate and fat content, either of which may be factors in the causation of frequent stools. A gastro-intestinal infection may be unassociated with fever and may therefore go unrecognized and be of serious consequence. Changes in the mother's environment, when she is nursing her own infant, as is seen at the end of the puerperium when she leaves her bed and changes her mode of living, seem to have a very beneficial influence on the quality of milk secreted, and the mother and baby seem to adapt themselves more readily to each other. This beneficial change is of course not seen where the baby is fed by a wet nurse.

At this point I desire especially to emphasize my experience with both mothers and wet nurses who are given a too liberal diet. While the average mother can be allowed to select her own diet during the nursing period of a full-term infant, eliminating such foods as may cause colic, abdominal distention and diarrhea in the infant, in the case of the premature such liberties must under no circumstances be allowed as they may result in an early disaster, cyanosis, and death due to abdominal disturbances. Therefore, every wet nurse should be made to adhere strictly to the limitations of diet as prescribed under the chapter on "Diet for Wet Nurses." So long as the infant is passing yellow stools of normal odor, without symptoms of indigestion and is gaining in weight, even though the stools may number five to eight daily, no alarm should be felt, and the diet should be sustained.

The change of the yellow stool to a green color shortly after passage is the normal process of oxidation. However, the green, frothy stool containing small white curds and considerable mucus should always be considered abnormal. Such stools usually lose their normal acid odor, cause excoriation of the buttocks, and are frequently associated with fever. This is not infrequently a finding in the breast-fed premature. In the treatment of such cases, while the care of the infant is of paramount importance, no less important is the careful regulation of the mother's surroundings, mode of living, and diet, and also her mental activity. Again every precaution must be taken in reducing the infant's diet, and the same dietetic measures which were instituted for the treatment of indigestion apply to every case of diarrhea with abnormal stools, as they are almost invariably attendant on an intestinal indigestion or infection.

Dehydration of the body tissues through excessive water losses must be met with sufficient water administration, so that a good working rule should lead one to administer at least one-sixth of the body-weight daily in fluids, in all cases of diarrhea. Therefore, when the quantity of the meals is reduced, or the interval lengthened, water should be added to the feedings or be administered between. The normal infant's stool will form a water margin about the semi-solid mass about one-half to three-quarters of an inch in diameter. When more water than this is lost with each stool, the infant must be carefully weighed and its water losses noted so that they may be compensated.

Tarry stools are always due to the presence of blood, and abrasions of the intestinal mucous membrane are likely to lead to fatal infections and must, therefore, always be given serious consideration.

Constipation.—The mechanical causes such as atresias in various parts of the intestinal tract, or an imperforate anus, must be considered as possible causes. As has been previously stated, diarrhea is far more common than constipation. This, however, does not mean that a sluggish lower bowel is uncommon in the premature. In fact the lack of power of the muscular wall and the minimal reaction of the mucous membrane to mechanical and chemical stimulation are both important etiological factors and are often associated with intestinal distention. The first evidence of this lack of response is often noted in the inability of the premature infant to evacuate the meconium which has accumulated in the lower bowel, and this may require mechanical removal by the aid of a small saline enema or further irritation with a soap or glycerine suppository. If these means fail, a single dose of five to eight drops of castor oil may be administered without too great delay, as it is our rule to start feeding only after the first intestinal evacuation, so that the presence of an atresia may have been noted, and the meconium removed before it has become infected through bacterial ingestion. The next stage of the infant's existence which is associated with constipation is in the first few days of life when food ingestion is insufficient and below the caloric requirements of the infant. In such instances in the absence of other causes it may be considered as a certain symptom of underfeeding, as is the case at all times when "hunger stools" are present. Increasing the food judiciously removes the trouble.

As the infant's digestive function improves and it utilizes its food to the fullest advantage, constipation may result from the minimal amount of food residue. The best evidence of such a causative factor in the presence of sufficient feeding is the improvement in general condition and gain in weight. In fact, utilizing their food perfectly, they have a tendency to constipation so long as their food intake is not in excess of their required caloric needs. Therefore feeding this class of infants moderately in excess of the normal caloric needs usually overcomes the constipation.

In the treatment where the ability to digest food is minimal, increasing the water intake is frequently beneficial. Even premature infants are creatures of habit, and where it is necessary to assist them in the evacuation of their bowels, this should be practiced at a stated hour, once or twice daily, either through the use of a saline or oil enema, non-irritating suppositories, or what is better, the tip of a well oiled catheter. Medication in the form of laxatives administered to the mother in the hope of influencing the character of the milk, as well as drugs administered directly to the infant are more or less dangerous agents and should be avoided whenever possible. Ten to fifteen drops of paraffin oil, 5 to 10 minims of milk of magnesia or equal amounts of castor oil, may occasionally be administered, but only after attempts at correction by mechanical irritation.

Underfeeding.—In order to consider this subject properly we must first take into consideration the types of infants with which we are dealing, that is, first, healthy premature infants, and secondly, congenitally debilitated infants, either premature or full-term. We must again divide them into the classes of breast-fed and artificially fed, and lastly, as to whether the underfeeding occurs during the first days of life during the period when normally we may expect stationary weight, or weight losses, or at later periods, during which we are more likely to see the development of the completed picture of marasmus.

Before entering upon the details of this subject several factors which tend toward the development of inanition and marasmus not directly dependent upon underfeeding must be considered. First and foremost is the danger of an imperfectly developed digestive tract, which even in the presence of sufficient food may soon result in a metabolic bankruptcy. These infants rarely survive the first days of life. Second, the improper hygienic surroundings of the infant, of which one of the most important is the danger of over-heating, thereby interfering with heat regulation and associated with excessive evaporation from the body surfaces. This is especially disastrous in its effects in the presence of decreased humidity. Both of these factors predispose to atrophy. Lessened immunity with the added dangers of local and general infections and the secondary nutritional disturbances are especially common in this class of infants, as are the tendencies toward repeated nutritional disturbances once they are established. All of these factors which may tend to impair the general nutrition of the infant will but serve to emphasize the need of careful observation. It will, therefore, be seen that underfeeding may be a primary affair, or may result secondarily following previous nutritional disturbances.

DISEASES PECULIAR TO PREMATURE INFANTS

Rachitis in Premature Infants.—The early appearance of rachitic manifestations in premature infants has been noted by many observers, especially associated with spasmophilia and anemia. Most prematurely born infants become rachitic—the lower the weight, the more certainly—and even human milk is not an absolute protection against this.

Huenekens* was able to collect 70 cases of prematures and twins, of which 58 developed definite signs of rachitis (82 per cent.). The time of occurrence was interesting, inasmuch as of 33 cases seen for the first time at or before four months 27 or 81 per cent. showed evidence of rachitis at that time. The first symptom usually noted was *craniotabes*, which in three instances was already present at six weeks. Langstein† observed it frequently in the third to the fourth month of life and in many a tendency to *convulsions* was noted due to hyperirritability of the nervous system of these infants.

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* Huenekens, Jour. Lancet, Dec. 15, 1917, xxxvii, p. 804.

† Langstein and Edelstein, Zeitschr. f. Kinderhlk., 1916, xv, p. 49.

Ylppö* observed commonly a *megacephalus* in connection with rachitis of the skull, which often left marks permanent for all life. These have to be regarded as characteristics of the prematures and not, as unfortunately is often the case, as signs of special "constitutional degeneration." Along with this megacephalus with its somewhat large, plump skull, there is asymmetry, which is not congenital but is produced in a mechanical way by the pressure of the infant's head in the first months of life and the softness of the skull.

The narrow thorax with its more or less marked signs of rachitis may also be regarded as a peculiarity characteristic of the smallest prematures but not of those of greater weight. This is not to be confused with the early functional, funnel chest which can be demonstrated in the first weeks of life and is due to the softness of the ribs in the smaller prematures. This leads to further deformity of the thorax, as the marked contraction of its lower half, which is the result of the congenital softness of the ribs and the rachitic affections later developing. The constriction around the chest is best seen about the insertion of the diaphragm.

The rachitic *rosary* is very prominent in prematures and is explained on the basis of the constant respiratory movements leading to deformities and marked enlargement of the epiphyses of the ribs.

The long cylindrical bones, however, only exceptionally show enlargements of the epiphyses in prematures, although rachitic changes appear in these bones very early. The process in these bones results rather in bone absorption and fringing of the epiphyses than in marked proliferation, which is the rule in strong full-term rachitic infants. The explanation of this feature in prematures may be in the fact that the rachitis appearing very early is already at end by the time the infant learns to walk, whereas in the full-term infant the hyperplastic epiphyseal enlargement occurs as a compensatory process in the period when the lower extremities are called upon to support the weight of the body. In the absence of special rachitic curvatures and epiphyseal enlargements of the long bones, we cannot, therefore, exclude rachitis in the premature. Histologic examination shows a characteristic picture in the absence of marked external manifestations.

The *etiology* of rachitis in premature and full-term infants has been the subject of much discussion. Huenekens believes that the explanation may be found in the abnormal chemical constitution of prematures. Their salt content is far below normal. Birk† found that a four months fetus contained 14 gm. of ash, at six months 30 gm., at nine months 100 gm., showing that two-thirds of the minerals were taken on during the last three months of fetal life. In the newborn fully 75 per cent. of this ash is made up of calcium and phosphate, the chief constituents of the bones. Huenekens believes, therefore, that the more premature the infant, the greater will be the deficiency of calcium and other minerals, so that by the third or fourth month of extra-uterine life the supply is entirely exhausted and rachitis results.

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* Ylppö, Zeitschr. f. Kinderhlk., 1919, xxi, p. 212.

† Birk and Orgler, Monatschr. f. Kinderhlk., 1910, i, p. 544.

Rickets in the premature may develop as a result of underfeeding. The inherited supply of calcium may be unduly drawn upon owing to the low calcium content of human milk and the difficulty of metabolizing sufficient quantities even of this food to prevent such over-draft. Severe rickets especially develops among the artificially fed, although the tendency is less when the diet contains an adequate amount of milk, rather than cereals or proprietary foods and a small amount of milk.

In a florid stage of rickets even though the calcium intake is ample there is a lessened calcium retention. Experiments with young dogs have shown that if the diet is deficient in other constituents even an abundant calcium content will not prevent rickets.

About 5 mg. per 100 c.c. is the average normal inorganic phosphorus concentration in blood serum. Howland and Kramer's investigations showed that the concentration of inorganic phosphorus in the serum was low in all patients suffering from rickets in the active stage, and that below the age of 2½ active rickets was found in all cases in which the blood serum showed an inorganic phosphorus content of 3 mg. or less. After the administration of cod-liver oil, as the healing process took place in the bones, the phosphorus content gradually rose to the normal. These authors concluded that when the serum of a young child showed a low percentage of inorganic phosphorus it was practically conclusive evidence that a condition of active rickets existed. They believe that calcium is not deposited, because of the phosphorus deficiency.

Infections also play a similar part. Impaired bodily functions directly affect the endocrine system causing secondary disturbances, so that while impaired mineral metabolism hastens the outbreak of clinical symptoms, still the secondary factors bear an important relation to phosphorus and calcium utilization.

Rickets therefore is a nutritional disturbance which affects especially the muscular and osseous systems, the resulting lesions preventing these structures from utilizing the calcium, and causing a diminution in its retention even though both blood and food intake are rich in it. The part taken by phosphorus in aiding the deposit of lime salt in bone is probably intermediary.

Treatment.—Our therapy is along the same lines as in full-term children with special stress on the feeding of human milk. Fresh air and sunshine in the older children and the observation of careful hygiene for all are without doubt highly important.

Diet must receive very careful consideration. In the very young the ideal food is of course mother's milk. Where artificial feeding must be instituted the amount of cow's milk should be minimal, and cereals and vegetables started early. Orange juice diluted with water should be given in small amounts from the second or third months (one to four teaspoonfuls daily). After the first month the diluent in the milk mixtures should be a cereal water (one tablespoonful of whole barley or oatmeal to the quart of water and not the dextrinized cereal flours). From the third month cereal and vegetable should be fed. The vegetable should be ground or cut into very fine pieces, boiled for several hours, and then fed in small quantities, beginning with half to a quarter of a teaspoonful three times weekly and gradually increasing. Spinach and carrots may be alternated, the secret of success seeming to lie in the fine division of the vegetable. After the fifth month vegetable soups should be given, substituting an ounce of soup for an equal amount of bottle feeding.

Cod-liver oil with phosphorus in a preparation containing 0.0003 gm. (gr. $\frac{1}{200}$) to each 4 c.c. (1 dr.) oleum morrhuæ, is a most practical mixture and can be administered to most infants by the fourth to the sixth week, beginning with 0.5 c.c. doses twice daily and increasing to 4 c.c. twice daily by the fifth month. The work of Schloss* has shown that the addition of a calcium salt to cod-liver oil with phosphorus further enhances the value of the mixture. Such a preparation is the tricalcium phosphate C.P. (10 per cent.) in emulsion of cod-liver oil U.S.P.

The Anemia of Premature Infants.—Closely associated with rachitis in premature infants is an anemia, which develops quite regularly and strikingly during the first three months of life. In our previous discussion of the physiology of the blood we noted from the work of Kunckel, Lichtenstein, Lande and others that in contrast with full-term infants, in the premature there is a greater number of nucleated red blood corpuscles, a more frequent appearance of myeloblasts and myelocytes during the first days of life, a lesser development of absolute and relative leukocytosis, and a greater number of immature leukocyte forms. There is also a distinct and very early hemoglobin impoverishment of the blood, which reaches its maximum in about the third to the fourth month.

Etiology.—Kunckel† believed that this anemia appearing regularly in the first three months of life was physiologic and was of the chlorotic type. His children improved in the second half year of life, but if infection was present at any time infants developed a severe secondary anemia much more readily than full-term infants. His opinion was that the anemia did not rest on an alimentary basis but was due to an insufficiency in hemoglobin metabolism beside a deficient iron storage.

Pfaundler‡ thinks that the anemia is closely related to a lack of fresh air and sunshine.

Lichtenstein§ fixes the early anemia in the first three months of life as a hypoplastic condition resulting through insufficiency of the hematopoietic system. The later oligochromemia, after spontaneous retrogression of the oligocythemia, he considers as a sequel of the improverished iron storage. He opposes the hypothesis of alimentary anemia of Czerny and Kleinschmidt. The theory of the harmful action of milk on the hematopoietic apparatus he asserts is disproved by the excellent results attending the feeding of human milk and the administration of small amounts of ferrous lactate.

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* Schloss, Zur Therapie der Rachitis, Jahrb. f. Kinderh., 1914, lxxix, p. 194.

† Kunckel, Zeitschr. f. Kinderhlk., 1915, xiii, p. 101.

‡ Pfaundler, Verh. d. Ges. f. Kinderhlk., Breslau, 1904, xxi, p. 24.

§ Lichtenstein, Svenska, Lokaresca as Kapets Handlinger, 1917, xlii, No. 4.

Lande* is in accord with the opinions of Kunckel and Lichtenstein. As evidence in favor of the importance of iron storage he emphasizes the fact that eighth month infants in the course of the second quarter year of life show a higher percentage of hemoglobin and erythrocytes than do seventh month prematures.

The examination by Lande of the bone marrow in 10 prematures disclosed no decisive picture except an insufficiency of the granulocyte system. Thus he disproves the theory that the basis of the anemia rests with a defective erythropoietic system.

Lichtenstein feels that there is no marked difference in the blood picture of artificially and breast-fed prematures. Examination of 28 cases artificially-fed, many of whom were born of nephritic, anemic and tuberculous mothers, showed no great differences in the blood picture from those breast-fed.

Symptoms.—The most marked symptom observed by Lande was pallor of the skin, which he saw with great regularity. It appeared especially early as a fore-runner of icterus, which in prematures is almost constantly present. The question arises as to whether the anemia is promoted by the icterus or both icterus and anemia are not bound up with a third factor—the maturity of the infant.

One can differentiate various grades of pallor, which is earliest and most clearly seen in the face and well portrays in general the degree of pathologic blood change. The most marked form of anemia gives the infant a bluish, transparent appearance, or a waxen, yellowish color, somewhat akin to the infants with severe congenital syphilis or chronic pyelitis. The picture is accentuated by the outstanding bluish veins, especially prominent on the skull and abdomen. The ears are transparent with hardly the vestige of a rosy hue and the mucous membranes are very pale.

The pallor after open-air treatment has been noted to give way to a rosy hue, but only in a few cases is there a parallel permanent increase in hemoglobin. However, with the increase in hemoglobin and erythrocytes in the fourth to sixth months, the color simultaneously improves.

In Lande's series the appetite of the infants was in general satisfactory. There was no stupor, especially in the more anemic. There were no elevations of temperature, as described by some observers, present with the marked blood changes.

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* Lande, Zeitschr. f. Kinderhlk., 1919, xxii, p. 299.

Marked glandular and splenic swelling was not observed by Kunckel or Lande, but Lichtenstein states that splenic tumor was present in two-thirds of his children.

Lichtenstein finds that the blood pictures in the well breast-fed prematures and those showing alimentary disturbances are both of the chlorotic type and differ mainly in degree. He also believes that the clinical picture described as pseudo-leukemic anemia is a severe form of secondary anemia and is not a distinct clinical entity.

Treatment.—For the general and hygienic treatment of primary and secondary anemia, the suggestions made for general measures in the care of rachitis should be followed. The infant must above all be given the advantage of a good environment, plenty of fresh air and sunshine.

Iron therapy for the purpose of increasing the iron content of the tissues and the hemoglobin has met with individual success. It should be started early. Among the iron compounds to be recommended are ferri carbonas saccharatus 0.25 to 0.5 gm., ferri et ammonii citratis 0.06 to 0.12 gm., or ferri lactas 0.12 to 0.25 gm. one to three times daily.

Small doses of liquor potassii arsenitis 0.03 to 0.06 c.c. may be given one or two times daily for short periods. The infant should be observed carefully for evidence of arsenic intoxication.

In the presence of congenital syphilis, mercurial therapy is imperative and may be combined with the arsenic treatment to good advantage.

It is of the greatest importance to bear in mind that as in the case of rachitis, the treatment for anemia should be started early. It is our custom to begin the prophylactic treatment of both of these conditions in the first weeks of life.

The Spasmophilic Diathesis in Premature Infants.—Besides anemia and rachitis, spasmophilia is one of the most interesting clinical peculiarities of premature infants. The term spasmophilia is used here in the sense of designating the manifestations occurring in the nursling, while tetany refers to the older child. Typical tetany with all its characteristic symptoms, the phenomena of Erb, Chvostek and Trousseau, carpopedal spasms, tonic and clonic convulsions, and laryngospasm, has rarely been observed in the premature newborn. While spasmophilia is by no means rare in artificially fed, full-term infants, it is found quite frequently in prematures, and not only in those artificially fed, but also in the infants fed on human milk. With breast feeding, however, spasmophilic manifestations occur only exceptionally and lead to convulsions usually only in connection with infections.

Etiology and Symptoms.—There are other predisposing factors besides feeding in the development of spasmophilia in prematures. We may not call these factors constitutional, since in all probability they depend on the various noxæ of the extra-uterine life. These noxæ lead to a hyperirritability of the nervous system, which in all premature infants is extraordinarily lowered during the first weeks of life against all possible stimuli, including the electrical.

On the other hand, however, it is a fact that feeding plays a very important rôle in the development of spasmophilia and above all in the appearance of convulsions. Not uncommonly, one observes prematures in whom the spasmophilic manifestations remain latent as long as breast feeding is continued, but appear shortly after the institution of artificial feeding. Langstein* reported a case of twins in whom convulsions always appeared shortly after artificial feeding was added to human milk. There were individual differences between the twins in the ease with which the convulsions could be produced. In one infant they developed within 7 to 12 days after the addition of artificial food, in the other within 18 to 20 days.

Rosenstern† studied the spasmophilic diathesis in premature infants and noted individual differences in the electrical hyperirritability, which appeared very early and frequently in breast-fed prematures. He was able to demonstrate spasmophilia in the form of electrical hyperirritability in 76 per cent. of the prematures or debilitated that he examined.

In Ylppö's series the electrical hyperirritability was not as frequent. Among the 42 premature infants in whom he was able to determine the electrical reaction systematically during the first three to six months of life, only 15 infants (35 per cent.) shows C.O.C. less than five milliamperes. In three cases electrical hyperirritability was already present in the second month. One infant was on human milk feeding and the other on mixed feeding. In the third month electrical hyperirritability appeared in three additional infants. It was most frequently present in the fourth month. From this data it seems that spasmophilia appears earlier in premature than in full-term infants.

Ylppö‡ also noted the especially interesting fact that the great tendency to electrical hyperirritability and convulsions, which he determined in many artificially fed premature infants, three to four months old, gradually disappeared in the fifth to sixth months without any treatment, while the feeding remained the same. In other children it often took months before the electrical hyperirritability disappeared.

In premature infants we frequently find very interesting deviations from the generally recognized symptoms of spasmophilia. It is not at all infrequent that the cardinal symptoms of spasmophilia (Erb's symptom), the electrical hyperirritability of the peripheral nerves, may be absent, in spite of the manifest signs of the disorder. To know this is very important, because we know that there exist conditions in premature infants in which the electrical reaction remains increased for months, although no convulsions occur. This lack of electrical hyperirritability in spasmophilic convulsions in prematures exists not only after convulsions have taken place—which could easily be accounted for by exhaustion of the nervous system—but also before the appearance of convulsions.

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* Langstein, Kassowitz, Festschrift, Berlin, 1912.

† Rosenstern, Zeitschr. f. Kinderh., 1913, viii, 171.

‡ Ylppö, Zeitschr. f. Kinderh., 1919, xxiv, p. 1.

In individual cases there may be pathologically increased electrical hyperirritability even when the electrical reaction does not go below five milliamperes for C.O.C. Rosenstern called attention to this fact and pointed out that the value below five milliamperes for C.O.C., which is regarded as pathognomonic for the spasmophilic diatheses, was determined by Mann and Thiemich* only for the age of eight weeks. From this it follows that this value is not to be regarded as a limit for younger infants, at least not for the younger prematures.

In the majority of cases the disappearance of the spasmophilic tendency in prematures occurs at the same time at which anemia and craniotabes begin to improve. Thus it becomes more and more apparent that the three symptoms, anemia, rachitis and spasmophilia are in a certain inter-relationship. It may very well be that the same harmful factors that damage the activity of the hematopoietic organs in the first months of life and also the growth and the normal calcification of the bones in such a high degree produce in some manner unknown to us changes in the nervous system.

Numerous investigators have demonstrated a calcium deficiency in the tissues, especially in the blood and the brain. Howland and Marriott have recently confirmed the earlier investigations, finding the calcium content of the blood to be low in this condition. According to their studies it averaged 5.6 mg. per 100 c.c. of serum in 18 cases in one group, the lowest being 3.5 mg. per 100 c.c. of serum, while the average normal is 10 to 11 mg. per 100 c.c.

There is, however, great question as to the relationship of parathyroid dysfunction and tetany in the infant. Pathological studies lead us to believe that parathyroid lesions in infantile tetany are the great exception. Parathyroid lesions have been described in patients who have shown no evidence during life of the pathognomonic findings of tetany.

In summarizing the pathogenesis we may state that a diminution of the calcium salts in all probability is the most important factor in the development of this condition. However, the possibility of an absolute or *relative* excess of the sodium and potassium salts, especially the phosphates, playing an important rôle, cannot be overlooked. The relationship of disturbance in parathyroid functions to the diminution of calcium tissue content must be made the subject of further study before its importance can be fixed.

Diagnosis.—The differential diagnosis of spasmophilic convulsions in prematures is very difficult. Among the conditions to be considered are hydrocephalus, congenital syphilis and tuberculosis, epilepsy, infections, brain injuries, asphyxia and pulmonary atelectasis. Tetanus neonatorum is rarely seen today. Meningitis and encephalitis are the most important of the infectious processes, the primary focus often being unknown. Perhaps the best test after careful history and physical examination is the determination of the electrical reactions.

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* Mann, Monatschr. f. Psych. u. Neurol., 1900, vii, No. 14.
Thiemich, Jahrb. f. Kinderh., 1900, li, 99 u., p. 222.

It must not be forgotten that given an injured brain and a marked tendency to spasmophilia this leads in the premature in the first place to convulsions and other manifest phenomena of this diathesis. There are only a few cases in the literature where special attention has been devoted continuously from birth to the later years to the condition of the spasmophilic infants. Ylppö's material enabled him to fill this gap to a certain extent. He was able to show positively that spasmophilia in premature infants very frequently occurred after a preceding injury to the brain. It is, therefore, not admissible to say without further evidence that spasmophilic convulsions produced the extensive damage to the brain.

Treatment.—The treatment of spasmophilia is largely prophylactic and embraces the therapy of rachitis and anemia (page 513). With the early institution and the continuation of these hygienic, dietetic, and medicinal measures, the development of spasmophilic convulsions will be very unusual.

If convulsions appear the infant must be kept absolutely quiet and warm. Narcotics are usually employed, the best being chloral hydrate, 0.25 to 0.5 gm. a day per rectum and calcium bromide, 0.5 to 1.0 gm. by mouth per day.

Lumbar puncture and withdrawal of 5 to 15 c.c. of spinal fluid may give relief from repeated convulsions.

The use of general anesthesia and morphine derivatives to control convulsions is to be avoided except as measures of last resort.

Magnesium sulphate in sterile 8 per cent. solution has been used in subcutaneous injections, 5 to 15 c.c. repeated once or twice within 48 hours, to control convulsions. Because of its marked depressive action on the nervous system, the infant must be very closely watched for collapse. Although this method has been much used in some clinics our experience has not warranted its use in preference to our preceding measures.

For the acute manifestations the calcium salts in maximal doses (preferably calcium lactate, 0.3 to 0.6 gm. three times daily, in solution or suspension) in our experience have been more valuable than the magnesium salts.

Administration of cod-liver oil and phosphorus or tricalcium phosphate in emulsion of cod-liver oil, as recommended in the treatment of rachitis, should be started at the same time and continued indefinitely, in doses varying from ½ to 4 c.c. twice daily, dependent upon the age and the indications.

When feeding by mouth is difficult, catheter administration must be resorted to together with inert fluids per rectum. If human milk is not obtainable the best substitute is albumin milk, which is poor in whey and rich in calcium. Where stimulating treatment becomes necessary that which has been previously mentioned may be employed.

In all our measures extreme gentleness must be used, as any rough handling or violence, in case of asphyxia, has a very harmful influence.

PROGNOSIS

In estimating the outlook for an infant born before the natural termination of the normal period of pregnancy, one must consider the prenatal and the post-natal factors before arriving at a conclusion. Of prenatal influences the most important is the absolute age; secondly, the physiological development and absence of constitutional anomalies; thirdly, transmitted parental conditions; and lastly, the presence of malformations. Of post-natal conditions the occurrence of any of the various diseases of the newborn affects the prognosis unfavorably as a rule, while the temperature and general behavior are of the utmost value in judging of its chances for life. In addition to these, the time at which the infant is received for treatment, and the character of the treatment it receives, go far in determining the probable outcome. While all factors must be taken into consideration, yet those of the most practical value relate to the child's behavior. Ability to nurse and swallow, coupled with strong muscular movements and a good cry, are the principal indications that the infant possesses a fair degree of vitality and resistance to disease, and that with proper care and nourishment it stands an excellent chance of resisting the enemies which threaten its existence during the first few weeks of its career, namely, cold and infection. At first doubtful, the prognosis becomes better as time passes in proportion to the care the child receives with respect to its hygiene and feeding. The secret of success in raising the premature lies in avoiding cold and infection, and in the proper selection of food as regards quality, quantity and method of administration.

There is not the slightest doubt but that the premature infant born of healthy parents, who is without congenital deformity and who survives the first few days of life, is entirely capable of complete and perfect development. The various factors that affect the outlook may now be considered in detail.

Age.—The prognosis of the premature infant depends in the first place chiefly upon the actual (fetal) age, or in other words upon the length of time it has remained within the uterus (Pfaundler); the infant born before the twenty-seventh week of pregnancy having but a slight chance of living. Other things being equal, those who are not too young can be raised.

The influence of the absolute age on the mortality is well shown by the figures of Potel:

Age	Number of children	Number dying	Per cent.
6½ fetal months	56	45	80.4
7 fetal months	131	76	58.1
7½ fetal months	53	17	30.1
8 fetal months	110	39	35.5

Sherman quotes the figures of several observers and includes those of his own experience in the Children's Hospital, Buffalo:

Incubator	Tarnier, per cent.	Charles, per cent.	Cook, per cent.	Gilbert, per cent.	Sherman, per cent.
Saved at 6 months	30	10	17	20	0
6½ months	..	20	20	..	..
7 months	63	40	50	35	50
7½ months	..	75	..	..	66
8 months	85	..	74	85	..
8½ months	95	..	..	..	100

Sherman's table shows the fallacy of the popular belief that more children are saved at the seventh month than at the eighth. All things being equal the older the premature the better its chance of life.

Great confusion exists in a study of various statistics because of the misapplication of the term "months;" the latter should apply to lunar months (28 days) and not calendar months or better the age should be stated in days or weeks to avoid all confusion.

Weight.—This is a much less dependable factor than age in estimating the outlook for the premature child. All conditions being equal, a small older child has a better chance of living than a younger one who weighs more. Nevertheless, a decrease in the death rate accompanies an increasing birth-weight.

Temperature.—In order to correctly estimate the power of resistance of a premature infant it is necessary to consider the degree of depression of the temperature and with it the weight of the child. The figures of Budin show that the lower the temperature the more serious any further reduction will be, and the less the weight, the more easily the child succumbs. Our experience has been that unless the child with subnormal temperature is soon placed in surroundings more favorable, the prognosis is grave. If, when placed in an incubator, the resulting rise of temperature is retarded, it is an unfavorable sign. An abrupt rise after a previously stationary hypothermia is also unfavorable. If the body temperature rises to 37° C. (98.6° F.) and remains there one may say that the nervous system is doing its work properly.

Porak and Durante estimated the lowest degree to which the body temperature may sink, with reparation still possible, as follows:

Infants with weight less than 1100 gm.	34°C.
Infants with weight 1100 to 1300 gm.	30°C.
Infants with weight 1300 to 1750 gm.	29°C.
Infants with weight above 1750 gm.	28°C.

Ylppö disagrees with the French observers in that his belief is that the mere cooling of the body surface does not result in death, but that many of these infants with subnormal temperature are the victims of birth injuries or brain hemorrhage, the latter factors resulting in fatality.

The Cause of Labor.—Generally speaking in these infants whose early birth depends upon the induction of labor, the outlook is better than when it results from spontaneous delivery. The following percentages are given as saved after induced labor:

Author	Per cent., born alive	Per cent., saved
Hahl	75.0	59.5
Raschkow	84.8	78.8
Heymann	....	71.2
Ahlfeld	....	90.9
Lorey	74.0	60.0
Hunziken	....	83.5
Ostreil	....	56.9

That there are many exceptions to the above statement cannot be doubted. For example: The occurrence of albuminuria may lead to the induction of labor, the child being not only premature but a weakling with low weight and vitality. On the other hand the infant born as a result of the shock attendant upon operative interference in non-suppurative appendicitis would in all probability possess excellent vitality. The artificial induction of premature labor with its associated trauma to the infant plays a very important part in the mortality. The foregoing figures are to be seriously questioned as there is no record of the birth-weight, which in many instances was undoubtedly well above 3000 gm. and, therefore, not strictly applicable to the premature infant.

Prenatal Influences.—The health of the mother during the period of gestation is of the utmost importance in prognosticating the immediate future of the premature and the weak. The occurrence of syphilis, tuberculosis, alcoholism, eclampsia, nephritis, severe heart disease, or other conditions producing faulty nutrition of the fetus, all have their effect on the well being of the infant. Of especial importance is the occurrence of syphilis or nephritis. Though necessarily the age and weight of the child have a direct bearing upon its physiological development yet the occurrence of constitutional diseases in the child is even of greater importance. Despite the greater age and the comparatively good development of a premature, the existence of a prenatal syphilitic infection or of an inherited predisposition to tuberculosis greatly jeopardizes the prognosis. If constitutionally well, the infant under 1000 gm. weight can live, providing sufficient attention is paid to the three conditions governing the survival of these infants. On the other hand, premature or even full-term infants, the victims of parentally derived diseases, often do not survive, regardless of the care they receive.

THE PHYSICAL DEVELOPMENT DURING CHILDHOOD

To be answered satisfactorily this question must be considered from several angles. First, the relation of mortality to the different age periods up to the time of puberty, and, second, the effect of premature birth on the mental and physical development of the child.

We may look for our best results during the first months of life in the premature born without physical defects, from healthy parents, and which has been able to take ample breast milk from its mother or a wet nurse. Similarly, in such an infant, we may expect a good response to artificial feeding when it becomes necessary to discontinue the breast. This group will also show a resistance to infection which compares favorably with that of a full-term infant. On the other hand, in the second and even later years of the defective infants, the congenitally luetic and those born to parents in poor health or late in life, more especially infants receiving little or no breast milk from birth, are likely to pass through a series of tumultuous years. The developmental and nutritional conditions prevalent during the first year will, of necessity, have a distinct bearing on the progress during early childhood.

The presence of early rickets and spasmophilia, together with the secondary anemia which so commonly accompanies these conditions, will leave their inhibiting impress upon growth and development during early childhood. As advised, prophylaxis against their appearance is imperative in well-directed care of prematures.

A distinctive pallor which tends to become progressive and is a peculiarly characteristic feature of the later part of the first year is probably due, to a considerable degree, to the disappearance of the fetal iron deposits and an inability to reestablish an iron balance on a diet low in iron.

It might be well in reviewing the life history of an average well-developed but small premature to consider the various changes in its physical appearance. Born with a slender primitive body, a round head, large in comparison with the rest of the body, rests on a thin and weak neck. The trunk is long, the chest flat, narrow above and broad below, giving a pyramidal appearance. The abdomen is broad and enlarged and unusually distended and showing a tendency to navel hernia. The arms and legs are thin and long and the fingers often reach below the knees. During this stage they present the "Old Man" type of appearance.

In the course of a few months characteristic changes appear. The slender primitive body becomes short and stocky owing to the gradually thickening neck and a general rounding out of thorax and abdomen. The head increases in size, the face is filled in by fat deposits and development of the muscular "sucking pads," in the cheeks. Exophthalmas is a common finding and the nose may lag in its development. The face assumes the "Doll" type. Although there is this increased growth in the head and trunk the rate of growth in the extremities remains retarded. This may be due to several factors, the most important being lack of use, with rickets playing a secondary role.

Many infants overcome their handicaps and make good progress, so that by the end of the second or third year their measurements compare favorably with those of the full-term child. A very considerable number (this is especially true of infants having a birth weight below 2,000 gm.) will display certain characteristics of body well into the school life. These features are as follows: The weight, length and thorax growth remains retarded, resulting in an apparent shortness of stature and general appearance of frailty. The skull, which may not be above the average, may still be out of proportion giving the appearance of a large head on a small, poorly developed trunk. The abdomen is usually protuberant and the extremities thin with large joints. The retardations up to this period, therefore, involve both length and mass of body. In most children, however, from the second or third year, there begins a gradual equalization, so that by the fourth to the sixth years the curves of growth run parallel with those of full-term children. Therefore in the presence of organs sufficiently mature to carry on the normal physiological requirements of circulation, respiration, metabolism, and excretion, and with nervous and endocrine systems sufficiently stabilized to coordinate the various body functions, normal body growth and mental development are to be expected.

Early defects gradually disappear, and in by far the larger percentage of cases of those who survive, the differences between the premature and the full-term child, have disappeared by the time of puberty. It is our belief that every perfectly developed premature infant should be given the advantages of proper environment, nursing and feeding care and every advantage needed to preserve its life.

THE MENTAL DEVELOPMENT DURING CHILDHOOD

While on the whole, in most cases, mental development progresses along parallel lines with physical development there are many exceptions which are to a large extent dependent upon the etiological factors underlying the premature expulsion of the fetus. Thus we do see a considerable number of these infants with good physical developments which show a retarded mental state, although the reverse is often experienced in that a frail body may house a precocious mentality.

It therefore becomes necessary in a general review of this phase of the subject to consider the infants as belonging in either one of two large groups: First, without known pathological changes, second, infants evidencing constitutional diseases or congenital malformation.

While a normal mental and physical growth is to be looked for in the first group, comprising well infants of viable age but immature, their progress must largely be dependent upon external influences. This means prophylactic feeding to insure a stabile nervous system, and the avoidance of all factors which may traumatize the nervous system, such as overstimulation in play, school, etc. Infections have a decided tendency to show an after effect on the nervous system.

Every effort should be directed to the prolongation of intra-uterine life, as the tendency to abnormal nervous manifestations is seen with lessening frequency as the fetal age increases.

In the second group, the congenitally subnormal premature and those with pathological changes in the nervous system secondary to birth injuries or post-natal conditions, any and all degrees of mental abnormalities are to be expected. Each case will of necessity differ in prognosis and indicated care. Thus there remains no other choice than to make the intra-uterine and extra-uterine noxæ responsible for the frequent cerebral disturbances, many of which are characteristic spastic states, with or without a tendency to convulsions. The mental development varies remarkably when one considers the possible pathological changes. I have several cases of spastic paraplegias and diplegias with good mentality, while on the other hand, the expected state of idiocy was present in other cases. I am certain that in at least a number of cases with good mentality the underlying pathology was gross intracranial hemorrhage. It is easy to conceive, in a given case with a flexible cranial cavity, that while a large subdural hemorrhage might cause some irritation it need not interfere with mental development. A small hemorrhage in the subtentorial or some other more confined region, however, might interfere with normal development of the brain and result in idiocy.

Fortunately only a small percentage of infants with gross pathological lesions are viable.

In summarizing our experience it may be stated that the majority of premature infants born after the thirty-second week into a proper environment, without birth injuries, undergo a normal mental development, progressing more slowly than the full-term infant during the first years. They average walking and talking about six months later and are somewhat slower in learning to coördinate, as evidenced by clumsiness and ease of falling, slight speech defects, etc. All of these are, however, usually temporary manifestations and are followed by normal progress.

A considerable number of prematurely born infants give early evidence of a neuropathic tendency as evidenced by the display of "temper spells" and other forms of emotionalism. These often follow in the wake of rickets, spasmophilia and secondary anemia, and are amenable to therapeutic procedures, the most important of which, in many instances, is a change of attendants. With proper environment, training and treatment of the underlying nutritional disturbances but little after-effect need be feared.

CHAPTER XXI

CHEMISTRY AND BIOLOGY OF MILK

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THE CONSTRUCTION AND FUNCTION OF MAMMARY GLANDS

Milk is the fluid secreted by the mammary glands and serves as food for the newly born mammal. The number of glands varies from 1 to 22 pairs in different mammals depending upon the number of young born in a litter. The mammary gland is located in the most convenient place for the young obtaining its product, and while most quadrupeds have these glands on the abdomen, in cattle the glands are situated in the region of the genital organs. In man and among apes the mammary glands are developed on the breasts; in some monkeys they occur near the armpits; in marsupialia the pouche is the site of convenience for the young; while the mammary glands of the female hippopotamus are located on her back in order that the young hippopotamus may feed while the mother is swimming.

The fully developed mammary gland is characteristic of the female, while in the male the undeveloped gland is present. There seem to be rare cases in which the mammary gland of the male has developed sufficiently to furnish milk for its offspring, but probably some reports of this phenomenon are legendary.

The anatomy of the cow's udder has been extensively studied and gives a clear idea of the gland structure. The cow's udder consists of two pairs of glands, thus forming four quarters and four teats. Bands of fibrous tissue separate each quarter, but this separation is complete only in walling off the quarters of one gland from those quarters forming the opposite gland.

The udder is formed of skin, muscle, nerves, veins, arteries, lymphatics, milk ducts, secreting glands, and fibrous, fatty, and connective tissue. The size of the udder varies considerably in different cows, the extremes being 24 to 54 by 16 to 32 by 10 to 20 cm. The soft skin of the udder is supported by fibrous tissue and is covered with delicate hair. During the process of milk secretion the udder swells considerably.

The outlets—teats—are tube-like appendages 6 to 10 cm. long. At the end of each teat is a sphincter muscle which prevents the loss of milk. In some animals however this sphincter muscle is weak and milk leaks out under the pressure of increased secretion. In other animals the sphincter muscle is so tight that dairymen have difficulty in obtaining milk and are obliged to insert a wooden wedge to keep the milk flowing. In extreme cases the muscle is weakened by a surgical operation. The skin of the teats has no sweat glands and no hair.

The cistern is a pouch separated from the teat by a circular muscle over which the cow has but imperfect control. The cistern holds one-half to one pint of milk. A network of milk ducts which become larger as they approach the cistern connects the cistern with other parts of the udder. They anastomose and finally form the ultimate follicles or gland lobules. Sphincter muscles under the control of the cow are located wherever milk ducts anastomose, the animal being able thereby to withhold milk, which is not an unusual occurrence when she is sick or dislikes the milker.

The secretory organs—alveoli—are located in the ultimate follicles. Three to five ultimate follicles are grouped together and have an outlet common with a milk duct. They develop during pregnancy and contract at the close of lactation. The follicles are lined with epithelial cells and are important factors in mild secretion.

The quantity of milk produced varies considerably in different breeds of cows, as well as in individuals of the same breed. Jersey cows may produce 6000 to 12,000 lb. of milk a year; Holstein-Friesians as high as 20,000 lb.; and Shorthorns average 6000 to 7000 lb. although the supply from a cow of this breed sometimes aggregates 10,000 to 12,000 lb.

The milk from different species of mammals varies materially in composition, but all kinds of milk contain protein, milksugar, fat, mineral matter, water and other substances of minor importance. The proteins are chiefly casein and lactalbumin. The casein in different milks is not the same, and while this is difficult to prove by chemical analysis, it has been shown by means of immunological tests, chiefly the precipitin and complement fixation tests, that caseins vary in different milks. Casein is a substance peculiar to milk and is classified among the phospho-albumins. It has never been found elsewhere in nature, not even in the body of the animal producing the milk.

Just how casein is formed in the mammary gland is a matter of conjecture. Some investigators hold that there is a special enzyme which forms casein from the proteins of the blood. Others believe that the proteins of the blood combine in some manner to form casein.

Lactalbumin is closely related to serum albumin and is possessed of similar physical and chemical properties but is different immunologically. Its formation in the mammary gland is obscure.

Milksugar or lactose is a disaccharid composed of dextrose and galactose. It occurs only in milk and has never been found elsewhere in nature, although the urine of lactating females sometimes contains it. As far as is known milksugar is the same in all milks, but it is uncertain by what process milksugar is formed in the mammary gland.

The fat in milk is the most variable component and is peculiar in its composition. All efforts to synthesize it so far have failed. It occurs only in milk and is not found elsewhere in nature.

The mineral matter in milk is the result of transformation of blood constituents in the mammary glands.

There is a common impression among producers of milk that the quantity and quality of milk can be materially influenced by feeding. In a certain sense this impression is based upon facts inasmuch as the general health of the animal affects milk production and milk quality. Careful experiments by a number of investigators have shown, however, that each species, each breed, and each individual produces milk of a certain composition and produces a certain maximum quantity, when in perfect health. Beyond definite limits no amount of food and no feeding of special kinds of food alters the milk. Under conditions, when cows are not properly fed or cared for, or when given unbalanced rations the quality and quantity of milk suffer, but with restoration of proper conditions the normal properties of milk reappear.

Since the quantity of fat in milk is commonly made the criterion of the quality, producers have attempted to "feed fat into milk" by means of oil cake and other foods rich in fat. This has not been a success. On the other hand, Jordan and Jenter have shown that a cow will, for a limited period at least, produce milk with normal fat content when fat was extracted from the food. Even the drinking of much water, or eating much succulent food does not influence the composition, according to Turner, Shaw, Norton and Wright. The fact, therefore, seems established that the composition of milk and the quantity produced are definite properties of certain breeds and individuals.

The difference in fat content in milk from different breeds is illustrated in the following table (Van Slyke and Publow):

THE PERCENTAGE OF FAT IN MILK FROM DIFFERENT BREEDS

Breed	Per cent., fat
Holstein-Friesians	3.26
Ayrshire	3.76
American Holderness	4.01
Shorthorn	4.28
Devon	4.89
Guernsey	5.38
Jersey	5.78

THE PHYSICAL PROPERTIES OF MILK

Fresh milk is an opaque, white or somewhat yellow fluid having a sweet taste and odor. Upon microscopic examination milk is seen to contain a large number of homogeneous discs which are strongly refractive, of a yellowish color and vary in diameter. These little bodies which appear to be structureless and without a membrane are fat globules. Microörganisms, cells and particles of foreign matter may also be present.

The fat in milk forms an emulsion; that is to say the globules are suspended throughout the fluid. When milk stands the fat globules rise to the surface because of the fact that the average specific gravity of milk fat is 0.93, while that of whole mixed milk is 1.027 to 1.034. The larger the fat globules the more rapidly do they rise to form the so-called cream layer. In this cream layer the largest fat globules are at the top and the smaller globules are found below, while the smallest ones, in cow's milk, remain suspended in the skim milk. Many microörganisms cling to the fat globules and rise with them, while others gradually settle. It is apparent, therefore, that the cream layer and sediment contain great numbers of microörganisms while the large intermediate layer contains relatively few.

The rising of fat globules in milk is influenced by several factors. In this regard the origin of the milk is a matter of much importance. In human milk, for example: separation of fat is rapid and fairly complete so that an opalescent fluid remains. In goat's milk, on the other hand, there is no spontaneous rising of fat unless the milk has been previously heated. In cow's milk the separation of fat is never complete, since some of the small globules remain suspended in the skim milk and creaming is retarded if the milk is heated. The reason for these differences is a matter of conjecture. Probably the size of the globules and the composition of the plasma are important factors. It is thought that the amount and colloidal condition of the casein are influential, inasmuch as increased casein content and large colloidal particles offer resistance to the rising of fat.

In fresh milk separation is more rapid than in old milk, and low temperature retards creaming. In shallow layers cream collects more rapidly than in deep layers, because the fat globules have less space to traverse.

Milk is a viscous fluid. This can be shown by allowing a definite volume to run from a pipette and comparing the time occupied in the process with that necessary to discharge an equal volume of water from the same pipette. The viscosity is not the same in all milk and the greater it is the slower is the separation of fat.

It has been mentioned that the large fat globules rise more rapidly than the small ones; therefore, milk with an average of large globules, as is the case with Jersey milk, separates more rapidly and more completely than milk with small globules. Centrifugation seems to retard creaming of milk.

Fat globules vary in size in cow's milk from 1 to 22 microns and the number per c.c. is from 1 to 11 million. A given sample of milk contains globules of various sizes, but the variation is greater in some samples than in others. For example: In goat's milk the extremes lie between 3 and 4.5 microns; in sheep's milk between 2.75 and 22.4; and in Jersey milk between 4 and 12 microns.

An emulsion is the result of several factors which are not yet entirely understood. It has been thought that the fat globules in milk are enveloped in a membrane which keeps them separated and suspended. The evidence cited in support of this theory is not entirely convincing. Others believe that there is an electric tension which keeps the globules in suspension, while still others contend that the colloidal casein is the means of keeping the globules in suspension. Further study on this point is desirable. The greatest difficulty encountered in determining the presence or absence of a membrane is found in the fact that some casein adheres to the globules even though they have been carefully washed in water, and what in reality may be adhering protein may be mistaken for a membrane.

Physical changes occur in milk during the period of lactation, especially in regard to the content of cellular elements and the size of the fat globules. Immediately after parturition, and for a period of about five days, the secretion of the mammary glands is a sticky, viscous and yellowish fluid termed colostrum. During this period the number of cellular elements is excessively large, so large that the protein content of colostrum may be as high as 15 per cent. or more, as compared with 3.5 per cent. in normal milk. The fat globules in colostrum milk are unusually large and frequently irregular in shape. The change from colostrum to normal milk production is so rapid that usually by the eighth or ninth day the milk is apparently normal. However, similar changes continue during the whole lactation period, although these are not so pronounced. The fat globules gradually become smaller and at the close of lactation are so small that cream does not rise readily and the making of butter is difficult.

The cellular elements in milk have given rise to considerable controversy concerning their function. Epithelial cells drop into the milk during milking; cells are constantly discharged from the secretory ducts; and leukocytes of all descriptions are usually present. The presence of various body cells has unfortunately given rise to the notion that they are derived from pus and the term pus cells is still sometimes used. Recent work, however, especially by Prescott and Breed, Russell and Hoffman and others, has shown that large numbers of cellular elements are frequently in milk of the highest quality. Information regarding the function of these cells must come from work to be carried on in the future.

The color of milk is due chiefly to the size of the fat globules, a pigment, and the colloidal casein. The larger the fat globules the more pronounced is the yellow tint in milk. The pigment, lactochrom, is contained in the fat and, if we are to accept the work of Palmer and Eckles, is composed of two parts. The more important component is carotin, a pigment widely distributed in green plants and grass. This accounts for the rich color of milk and butter when the cows graze on green pastures in early summer. The whiteness of skim milk demonstrates this fact, and further substantiation may be observed by dissolving pure fat-free casein in alkaline earths. Such a solution is white and opaque, similar to skim milk. The white color of milk is due to colloidal casein.

Clean fresh milk has a slightly sweetish taste and agreeable odor. The "cowy" taste and odor not uncommon in milk is the result of manurial pollution and absorption of odors from the air.

The viscosity of milk changes with temperature. Cold milk has greater viscosity than warm milk, while heated milk loses much of its viscosity. This can be restored by the addition of a small quantity of viscogen prepared by mixing a concentrated solution of cane sugar with freshly slaked lime. After standing the clear solution is poured off and mixed with 100 to 150 parts of milk.

The specific gravity of cow's milk varies between 1.027 to 1.034 at 15°C. Human milk and the milk of goats have a similar specific gravity. The dissolved substances in milk are responsible for increased specific gravity, while the fat tends to lower it. So-called rich milk, therefore, has a lower specific gravity than milk poor in fat; skim milk has a higher specific gravity than whole milk, while cream shows a lower specific gravity than either whole or skim milk.

The freezing point of milk is 0.54 to 0.57°C. below that of water. When milk freezes important physical changes occur which cannot be entirely obliterated during the process of thawing. As freezing commences from the outside, the solids are forced towards the center and a more highly concentrated solution with a lowered freezing point results. Examination of layers of frozen milk shows that the solids are smallest at the periphery while highest in the center. The fat moves not only to the center but also to the top.

THE CHEMISTRY OF MILK

The composition of milk from different species of mammals varies greatly as shown in the following table:

Table 1.—The Composition of Milk from Different Mammals
(Compiled from Different Sources)

Animal	Specific gravity	Water	Casein	Albumin	Total protein	Fat	Sugar	Ash	Total solids
Man	1.0298	87.58	0.80	1.21	2.01	3.74	6.37	0.30	12.42
Cow	1.0313	87.27	2.88	0.51	3.39	3.68	4.94	0.72	12.73
Cow colostrum	1.042	75.07	4.19	12.99	17.18	3.97	2.28	1.53	24.93
Ass	1.032	90.12	0.79	1.06	1.85	1.37	6.19	0.47	9.88
Sheep	1.0355	83.57	4.17	0.98	5.15	6.18	4.17	0.93	16.43
Goat	1.0305	86.88	2.87	0.89	3.76	4.07	4.64	0.85	13.12
Mare	1.0347	90.58	1.30	0.75	2.05	1.14	5.87	0.36	9.42
Reindeer	......	67.00	8.30	1.50	9.80	17.00	2.80	1.50	33.00
Rabbit	1.047	69.50	....	....	12.00	13.50	2.00	2.50	30.50
Dolphin	......	41.00	....	....	11.00	46.00	1.30	0.60	59.00
Sow	1.038	83.94	....	....	7.23	4.55	3.23	1.05	16.06
Bitch	1.035	75.44	....	....	11.17	9.57	3.09	0.73	24.56
Elephant	1.0313	79.30	....	....	2.51	9.10	8.59	0.50	20.70
Camel	1.042	86.57	....	....	4.00	3.07	5.59	0.77	13.43
Llama	1.034	86.55	....	....	5.05	7.51	4.44	0.75	17.75

The table shows a variation in casein of from 0.79 in ass' milk to 8.30 per cent. in reindeer milk; of protein from 1.85 in ass' milk to 17.18 per cent. in cow colostrum; of fat from 1.14 in mare's milk to 46 per cent. in dolphin's milk; of sugar from 1.30 in dolphin's milk to 8.59 per cent. in elephant's milk; of ash from 0.30 in human milk to 2.50 in rabbit's milk; and of total solids from 9.42 in mare's milk to 59 per cent. in dolphin's milk. The greatest variation is seen to be in fat content.

The compounds contained in milk may be grouped as follows:

1. Fat
2. Protein
3. Carbohydrate
4. Mineral matter
5. Water.

Besides these main groups there are other substances, namely, lecithin, extractives, cholesterin, orotic acid, pigments, citric acid, lactomucin, and gases, but these substances are of minor importance as compared with the main groups. In addition enzymes and immune bodies are invariably present in fresh milk.

It it sometimes stated that amino-acids, ammonia, hydrogen sulphide, marsh gas, carbohydrates—besides lactose—and other substances have been detected in milk. Whether these substances are genuine milk constituents, or the result of microbial action, or byproducts of chemical analysis is not determined. The evidence of these findings is not conclusive and it is quite probable that they are due to chance.

Sometimes milk contains volatile substances derived from certain aromatic foods, but it is not definitely known whether these substances pass through the mammary glands or are absorbed by the milk after milking. Easily soluble substances, however, such as medicines, etc. have been known to pass through the mammary glands and into the milk from which they may be recovered.

The most important proteins in milk are casein and lactalbumin. There is also a globulin present and other proteins have been reported to be present, but the latter may be the result of microbial activity and not inherent milk proteins. Van Slyke gives the following as the average composition of cow's milk:

TABLE 2.—ANALYSIS OF COW'S MILK

	Water = 87.1			
	Solids = 12.9		N compounds = 3.2	Casein = 2.5
	100.0	Fat = 3.9	Milksugar = 5.1	Albumin = 0.7
Milk = 100		Solids-not-fat = 9.0	Ash = 0.7	3.1
	CO_2	12.9	9.0	
	Gases = N			
	O			

Colostrum differs from normal milk in its greater albumin content, its higher ash, and lower fat content. The percentage of casein and milk-sugar is less than in normal milk.

When the casein is removed milk plasma remains, and has of course, a lower percentage of total solids than cream or whole milk. When cream and casein are removed a translucent yellowish fluid, the whey, remains. This contains water, milk-sugar, albumin, mineral matter and a variable remnant of fat, amounting to 0.3 to 0.5 per cent. The composition of whey is given by different analysts as follows:

TABLE 3.—THE COMPOSITION OF WHEY

Analyst	Water	Fat	Milk-sugar	Protein	Ash
Van Slyke and Publow (cheese) factory milk	93.04	0.36	5.76 (+ash)	0.84	
Fleischmann	93.15	0.35	4.90	1.00	0.60
König	93.38	0.32	4.79	0.86	0.65
Smetham	93.33	0.24	5.06	0.88	0.49
Vieth (from skim milk)	93.00	0.09	5.45	0.92	0.54

The chief difference between buttermilk and skim milk is the presence of acid in the former. Lactose decreases under the influence of bacterial action and acid, chiefly lactic acid, is formed.

THE CHEMISTRY OF BUTTER-FAT

Butter-fat is composed of triglycerids, cholesterin and a pigment. It is probably a complex compound and not a mere mixture of constituents. If it were a physical mixture the compounds of low melting point would melt before those of high melting point, while as a matter of fact butter melts uniformly. The tributyrin contained in milk-fat has a bitter taste and would impart its peculiar taste to the butter if it were present in its normal form, but it cannot be extracted from butter even with alcohol although it is easily soluble in it. If milk-fat is heated to 275°C. there is no distillate, therefore, the volatile acids must be present in combined form. Finally, an artificial mixture of the compounds known to be present in butter-fat has a higher melting point and a higher molecular weight than natural butter-fat. Of course, butter-fat may not be a single compound, but it is reasonably certain that the compound is more complex than the substances isolated.

According to Richmond the glycerids of volatile acids form 17.3 per cent. of milk-fat; according to Van Slyke 8.5 per cent. The most important glycerids are butyrin, caproin, caprylin, caprin, laurin, which are glycerids of voaltile acids, and myristicin, palmitin, stearin and olein, glycerids of non-volatile acids. The aroma and odor of butter are due chiefly to volatile acids.

Butter-fat melts at 29.5 to 36.0°C. It is liquid when milk is warm, but solidifies when milk is cooled. The spherical shape of the fat globules becomes irregular when milk-fat solidifies.

Milk-fat is soluble in ether, carbon disulphide, nitrobenzene and acetone. It is slightly soluble in ethyl alcohol, sparingly soluble in cold amyl alcohol, but completely soluble in hot amyl alcohol.

Milk-fat may decompose under the influence of light, oxygen, and some types of microörganisms, and becomes rancid in taste and odor.

THE CHEMISTRY OF CASEIN

Since the casein of different kinds of milk varies in composition, it is proper to speak of "caseins" rather than of "casein." Bovine casein is weakly acid and occurs in milk in combination with calcium. It is spoken of, therefore, as calcium caseinate and is a tri-basic acid. When milk is filtered through a porous filter the casein splits into two components, a soluble portion which passes through the filter and an insoluble portion which remains on the filter. Casein is in colloidal solution and the particles can be seen when milk is examined with an ultramicroscope. Casein is also removed from milk by centrifugation and thrown into the bowl sediment.

Small quantities of dilute acids precipitate the casein in fine flakes, while large quantities produce a compact curd. Excess of acid redissolves casein. Pure casein which is used for many commercial articles is prepared from skim milk by precipitation with acid, redissolving in alkali and reprecipitating.

Pure casein is a tri-basic acid which replaces CO_2 in carbonates. It is insoluble in water and alcohol, soluble in acids when not highly diluted, in alkalies, alkaline earths, and alkaline carbonates. It yields no ash and has never been crystallized.

Elementary analysis of bovine casein is, according to Richmond, as follows:

TABLE 4.—FORMULÆ AND COMPOSITION OF CASEIN

	$C_{162}H_{258}N_{41}SPO_{32}$, per cent.	$C_{170}H_{268}N_{42}SPO_{51}$, per cent.
C	52.96	54.04
H	7.03	7.10
N	15.64	15.56
S	0.86	0.84
P	0.84	0.82
O	22.41	21.60

Caseins from different milks have different combining powers; different electric conductivity; and produce different antibodies when injected into animals. Caseins of milk from solipeds are more readily soluble than caseins from ruminants. All caseins contain phosphorus.

Bovine casein is white, amorphous, hygroscopic, tasteless and odorless. The specific gravity is 1.259. It gives the protein reactions, excepting the Adamkiewicz and Mölisch reactions, and furnishes 5742 calories per gram. Van Slyke and Bosworth give the molecular weight at 8888 and that of paracasein at 4444. Alkaline solutions of bovine casein are levorotatory. Casein contains no carbohydrate radicle; glycocoll is absent and cystin present in small quantity. Tyrosin and mono-amino-acids are present in fair quantity. Casein is precipitated by the salts usually employed for protein precipitation, chiefly the sulphates of ammonium and magnesium.

A very interesting and not yet fully understood reaction is the coagulation of casein by rennet. Rennet produces different results in milks from different species. In human milk, for example, rennet coagulates the casein only when acid is added, while cow's milk is readily precipitated. According to Van Slyke and Publow rennet coagulation occurs in three phases: First, the casein is changed into paracasein; secondly, insoluble calcium salts are changed to soluble calcium salts; and thirdly, the uncoagulated paracasein is precipitated by soluble calcium salts. The coagulation of rennet is accelerated by suitable distribution of the rennet; by a temperature of 41°C.; and by a weak solution of the salts of alkaline earths. A slightly acid reaction is favorable to rennet action, but strong or concentrated acids retard. High and low temperature, free hydrogen ions, presence of formalin, high dilution of rennet, or concentrated salt solutions retard rennet action.

LACTALBUMIN

Lactalbumin is similar to, but not identical with, serum albumin and can be prepared by heating the filtrate remaining from casein precipitation. It is crystallizable and soluble in saturated solutions of sodium chloride and magnesium sulphate. It is precipitated by a saturated solution of ammonium sulphate and by the addition of acetic acid to a saturated solution of magnesium sulphate. The influence of heat applied to lactalbumin was found by Rupp to be as follows: There is no coagulation at 62.8°C. for 30 minutes; at 65.6°C. 5.71 per cent. is coagulated; at 68.3°C. 12.75 per cent.; at 71.1°C. 30.87 per cent. Coagulation, therefore, is not complete at 70°C.

LACTOGLOBULIN

There is a small amount of globulin in milk which is probably identical with serum globulin. This globulin is soluble in sodium chloride solution, is precipitated by magnesium and ammonium sulphate and by tannic acid, but is not coagulated by rennin. It coagulates at 72°C. Colostrum contains a larger quantity of globulin than normal milk. It has been thought that lactoglobulin carries the immune bodies present in milk and colostrum therefore is richer in antibodies than normal milk.

MILK-SUGAR

Milksugar, or lactose, is probably the only carbohydrate in milk. There is no reliable evidence that other carbohydrates are inherent components, excepting perhaps the carbohydrate "tewficose" isolated by Richmond from the milk of the gamoose or the Egyptian buffalo.

Milksugar is less soluble in water than canesugar and dextrose, and its solution is, therefore, less intensely sweet. It crystallizes in rhombic prisms enclosing one molecule of crystallization water.

THE MINERAL CONSTITUENTS OF MILK

The mineral constituents of milk are usually given as ash, but this of course does not give account of the salts actually present. Some of the organic phosphorus appears in the ash while inorganic parts may volatilize. The quantity of ash in different milks differs. Goat's milk, for example, contains 0.7 per cent.; mare's milk 0.28 to 1.2 per cent.; ass's milk 0.3 to 0.4 per cent.; human milk 0.19 to 0.34 per cent.; and cow's milk 0.7 to 0.9 per cent.

The salts of potassium, sodium, chlorine, citric acid, some phosphates, calcium salts and magnesium salts are in solution; the di- and tri-phosphates are in suspension. Edelstein and Czonka found 0.4 to 0.7 mg. of iron per liter of milk. The salts are probably derived from food and water and are essential for the food value of milk. The quantity and kinds of salts are changed by the loss of carbon dioxid, heating, formation of acid, diseases of the animals, food, lactation period and other conditions.

GASES IN MILK

Normal milk contains oxygen, nitrogen, and carbon dioxid. Fresh cow's milk contains seven to nine volume per cent. of gas, while in human milk there is but one-tenth this amount. As milk stands in contact with the air there is a gradual exchange of gases until the gas content of the milk resembles that of the air. Gases, however, are removed in large measure by the application of milking machines. Marshall thinks that part of the carbon dioxid present in milk is loosely combined, since he had difficulty in removing the last traces.

Milk is very susceptible to absorption of gases in the air. Consequently foul smelling gases of the stable are easily dissolved in milk and it is, therefore, customary in many dairies to aerate the milk before bottling. Milk produced in clean stables does not need aeration.

THE REACTION OF MILK

The chemical reaction of milk is due chiefly to the salts which are present. Acidity is due to acid phosphates, citrates and casein, while alkalinity is caused by alkaline phosphates. As a rule, milk from carnivorous animals is more acid than milk of herbivorous animals. Most kinds of milk react acid to phenophthalein and rosolic acid, and alkaline to dimethyl-orange.

Milk is amphoteric to litmus. Since carbon dioxid is in part the cause of acidity of cow's milk, the reaction becomes less acid for a short period after milking, owing to the escape of carbon dioxid. Later, under the influence of bacterial activity the amount of acid rapidly increases. Towards the close of lactation the reaction is usually somewhat less acid than at the beginning.

VARIABILITY IN THE COMPOSITION OF MILK

The composition of milk varies not only with the species from which it is derived, but also with breeds and individuals of the same species. Even milk from the same animal is subject to some variation. These variations may be grouped as follows: Difference in composition due to breed; difference due to individuality; difference of milk from the same individual; difference during the lactation period; difference due to food; difference due to season; difference due to the skill of the milker; difference due to the health of the animal.

Difference in composition due to breed: The fat content is highest in Jersey milk while that of Guernseys follows closely. The milk of Ayrshires, Shorthorns and Devons occupies an intermediate position, while that from Holstein-Friesians contains the smallest amount of fat. The casein content shows a similar variation, but the difference is not nearly so great as the difference in fat content.

As to the size of the fat globules, Jersey milk has the largest globules, followed by that of Shorthorns, Ayrshires and Holstein-Friesians in the order named.

There is little difference in the amount of lactalbumin in the different breeds.

Difference in milk from the same individual: There are variations in the composition of milk of different milkings; differences between morning and evening milk; and between foremilk, middle milk and strippings. While composition of milk from different milkings and of morning and evening milk does not vary greatly, the fat content of the first streams of milk, of the middle milk and the strippings shows considerable variation. As a matter of fact the strippings may contain 10 times as much fat as the foremilk. There are several explanations of this phenomenon offered: First, the simple, although not entirely satisfactory explanation, that the fat globules rise in the udder in a manner similar to that in a bottle; second, and what appears to be a more plausible explanation, is the mechanical adhesion of the fat globules to the walls of the milk ducts from which they are removed only by persistent milking.

The plasma solids of foremilk and strippings do not differ appreciably.

Variation in composition during the lactation period: During the period of lactation there is a progressive change in the composition of milk. This change has been extensively studied and the results are fairly uniform. Lactation commences with the appearance of colostrum milk which rapidly passes into normal milk. The quantity of milk produced diminishes as lactation progresses, at first slowly, but quite rapidly towards the close of lactation. The solids, however, after an initial decrease for a few weeks increase slowly up to the eighth or ninth month and during the last weeks of lactation increase rapidly. The food value of milk, therefore, increases constantly. Fat is the most variable constituent, but the proteins also vary in large measure. The milksugar and ash do not increase materially.

The size of the fat globules, as previously stated, decreases as lactation advances, but the number of globules increases. At the close of lactation all globules are small, but sufficient in number to actually increase the percentage of fat.

Influence of Season and Weather on the Composition of Milk.—There is a seasonal variation in the composition of milk, although observations on this subject do not entirely agree. While Van Slyke and Publow state that the greatest production of milk and fat commences about the middle of May, Eckles and Shaw claim that the largest amount of fat produced is in the autumn and early winter.

Trunz in his studies of the mineral content of milk states that the ash content is lower in spring and summer than in winter.

In regard to weather changes there is probably no variation in the composition of milk from this source, but it stands to reason that cows should be protected against inclement weather as a measure of preserving their health.

Variation of Composition of Milk Due to the Skill of the Milker.—The influence of the milker on the flow of milk is twofold. First, it has been shown that the strippings contain a much higher percentage of fat than the fore- and middle-milk, hence, the milker who is able to extract the last portions of milk from the udder will obtain a richer milk than one who does not do so. Second, it is probable that a psychological element is involved, since it seems that the cow will yield her milk more readily to the milker who treats her kindly or to whom the cow is accustomed, than to a milker who ill-treats the animals.

Changes in Composition of Milk

While the milk is in the udder there are few, if indeed there are any, changes in composition, but as soon as it has left the udder changes become manifest. The chief physical changes consist in the rising of fat and the discharge of gases, while chemical changes are profound and in large part due to the action of microörganisms. The chief sources of change in composition are: Spontaneous changes; application of heat; centrifugation; dialysis; electricity; microörganisms and enzyms.

Spontaneous Changes.—It has been mentioned that there is an exchange of gases with the air when milk stands. Oxygen and nitrogen are absorbed, while carbon dioxid is discharged. The specific gravity of milk is increased by the loss of gas and the acidity is reduced by the loss of carbon dioxid. The loss of carbon dioxid, furthermore, causes precipitation of some phosphates by changing them to insoluble compounds. This precipitation is especially pronounced when milking machines are used as these depend upon suction which removes carbon dioxid gas in larger measure than does mere contact with the air.

The creaming of milk, which has been previously discussed, is also a spontaneous change. If milk freezes there are profound physical changes. These also have been described.

Changes Due to Heating Milk.—The changes due to the heating of milk are of particular interest since heat is applied quite generally to preserve milk and to destroy microörganisms. The changes due to heat become more pronounced with the greater degree of heat applied and the length of time of exposure to the heat.

Practically all constituents of milk are influenced by heat: The casein is altered; albumin is precipitated (see page 567); fat globules coalesce; some of the salts are precipitated; gases escape; the milk acquires a peculiar taste, the so-called cooked taste; and the yellow color deepens. When milk is heated in contact with the air a film forms which becomes harder with the application of higher degrees of temperature.

The film, sometimes called "haptogen membrane," begins to form at 40°C. at which temperature it is soluble in lime water. At higher temperatures it becomes insoluble in lime water. It is composed of an insoluble variety of casein, caseid, and also contains some of the other constituents of milk.

Colostrum milk which is rich in albumin coagulates when boiled because of the precipitation of albumin. At 80°C. the casein is changed so that rennet does not coagulate the milk promptly, and the higher the degree to which milk is heated the greater becomes the soluble portion of casein. While in raw milk but 6.41 per cent. of protein is soluble, in milk heated to 140°C. for one hour 25.61 per cent. is soluble.

Milksugar is not changed materially by boiling, but begins to decompose at 120°C. and caramelizes with the subsequent formation of acids.

The gases are almost entirely removed by boiling so that through loss of carbon dioxid the milk becomes more alkaline than raw milk.

Fat is not decomposed at the boiling point or even by higher temperature. The physical condition of the fat, however, commences to be altered at 70°C. or below. The fat clusters are broken up and small globules are liberated so that creaming becomes slow and is less complete. This process is hastened by higher temperature and by longer exposure to a certain temperature.

The "cooked taste" is, in large measure at least, due to protein decomposition by which hydrogen sulphide is liberated. The casein of boiled milk, when precipitated by acids, forms smaller flakes than that of raw milk, hence casein from boiled milk is more easily digested than that from raw milk.

Changes in milk produced by centrifugation.

Since a large amount of milk is centrifugalized, partly for the removal of cream and partly for the removal of foreign substances, it becomes important to know whether any changes in composition are caused by this process. It has been found that the insoluble parts of milk, excepting fat, are thrown into the bowl sediment. Suspended phosphates are largely removed and the ash of the bowl sediment is, therefore, relatively large. The colloidal casein is also partly thrown into the sediment and the quantity increases with continued centrifugation. Insoluble dirt, microörganisms and cellular elements are thrown into the bowl sediment.

Dilution of Milk and Dialysis

By diluting milk the reaction is changed so that less alkali is required for neutralization. By dialysis the casein is precipitated due to the loss of soluble calcium salts.

The Influence of Electricity on Milk

It is frequently stated that thunderstorms cause milk to decompose readily, but there is no scientific evidence to support the assumption. It is possible, however, that the heat and moisture usually accompanying electric storms cause the temperature of the refrigerator to rise so that bacteria grow and decompose the milk. It is possible, furthermore, that the effect of a moisture-laden atmosphere affects the men handling the milk so that proper care is not always exercised, with the result that more bacteria enter the milk than ordinarily. Passing electric currents through milk, however, has not increased its liability to decomposition.

The influence of microörganisms and enzyms on the composition of milk is discussed elsewhere.

ADULTERATION OF MILK AND PRESERVATIVES

Adulterations of milk are not as commonly met with as formerly and preservatives are rarely used. In former times a pump was a necessary adjunct to the cow stable so that cans of milk could be filled with water when the milk supply was insufficient. The supervision of milk supplies by health departments has largely done away with such abuses.

Still it is desirable to test milk to learn whether it has been diluted with water, skimmed, or whether preservatives have been added, because a relaxation of control would probably encourage abuse.

The food value of milk may be reduced by the addition of water, by skimming, or by both.

By adding water to milk all constituents are diluted, and when this is done to reduce a high fat content in order to bring the milk within legal limits the other solids are proportionally reduced. As the plasma solids, or solids-not-fat, are fairly uniform in all kinds of cow's milk, dilution can easily be detected, at least a dilution with 10 or more per cent. of water. Skimming of milk can be detected by determining the fat content. However, skimming is practiced chiefly on milk with high fat content and then escapes detection. When both skimming and watering are practiced the low fat content and low percentage of plasma solids are indicative.

Sometimes thickening agents are added to increase the body of milk, especially when water has been added, or fat removed. Gelatin is sometimes dissolved in buttermilk, and cane sugar is added to sweet milk to increase the plasma solids and cover up acidity. However, these additions are relatively scarce.

Dilution of milk with water is to be condemned not only because it is fraudulent but because it involves the possibility of using polluted water supplies through which diseases may be spread. The addition of sucrate of lime or gelatin for increasing the viscosity of milk is not objectionable from a sanitary point of view, but milk treated in this manner should be suitably labeled.

Foreign fats are sometimes added to milk in order to increase its richness. It is difficult to detect these by chemical means and when such milk is forced through a homogenizing machine the added fat cannot be detected by microscopical examination. The addition of foreign fat may increase the fat content, but adds nothing to the aroma of milk since that is peculiar to milkfat.

Coloring matter is added to milk in order to give it a rich appearance. Annatto, caramel and anilin dyes are used for this purpose. Annatto and caramel are harmless deceptions, but anilin dyes may contain poisons. Annatto enters into the fat, while caramel and anilin dyes also color the skim milk. By precipitating the casein the presence of caramel and anilin dyes can easily be detected, since they leave the casein colored, while annatto remains in the fat leaving the casein white.

Preservatives are added to milk to enhance its keeping qualities through the destruction of microörganisms, or to neutralize the acid in partially decomposed milk.

Formalin in some form or other is the commonest preservative used, but can be detected in very small quantity. Formalin is sold under various trade names to hide its identity and is frequently used without the milk dealer's knowledge. Besides formalin, boric acid, borax, salicylic acid, benzoic acid and perhaps other substances have been used, but are found rarely at the present time.

Formalin causes a distinct reduction of germ content when added in a proportion of 1:10,000 to 1:20,000. In a dilution of 1:40,000 the reduction is less pronounced. Whether it is harmful in such quantities is an unsettled question. According to some investigations infrequent consumption of formalin in these amounts seems to have no untoward effect, but whether continued ingestion would be injurious is a different question. At any rate there is no necessity for the use of preservatives of any sort, since milk produced under sanitary conditions as it should be keeps long enough to satisfy the consumer.

There is a further menace in the application of preservatives due to the fact that they may be added to the milk at different stages of handling. The producer may add a preservative, more may be added at the collecting station, and finally the dealer may add a third quantity causing an accumulation of preservative in the same milk sufficient to injure its healthfulness.

ENZYMS IN MILK

In the literature the term "life in milk" is sometimes encountered, but the term is not always used in the same sense. While some authors conceive a vague sort of notion that the milk constituents are endowed with a property similar to life which renders them more suitable for infant feeding than other "dead" foods, the majority apply the term "life in milk" to the presence of enzyms. The latter group of authors distinguish between "inherent" milk enzyms, by which they mean enzyms naturally present in milk, and enzyms which are excretions of microörganisms. After studying the evidence for this distinction it must be confessed that there is considerable difficulty in clearly separating the two groups of enzyms. Microörganisms are present in milk even in the udder and it is, therefore, difficult to determine the real origin of enzyms. It is true that some authors claim that they can obtain sterile milk by introduction into the udder of a sterile tube connected with a sterile flask. Most authors, however, believe that even the strippings contain some bacteria and, admitting the possibility of obtaining sterile milk from the upper parts of the udder, the quantity obtained is of necessity very small for investigational work.

Babcock and Russell have attacked the question from another angle. By the addition of chloroform or toluol they prevented bacterial growth in milk and still observed distinct enzym activity. But here again the question is, were the enzyms formed previous to the addition of a preservative or afterward. For the authors did not work with milk entirely free from bacteria, although it was obtained under stringent precautions to keep the number of microörganisms low. It is, therefore, impossible in the present state of our knowledge to state whether "inherent" milk enzyms exist or not. But it is known that enzyms are always present in unheated fresh milk.

Investigators have found proteolytic, carbohydrate-lytic, oxidizing and reducing enzyms in milk. The proteolytic enzyms have been considered of importance in cheese ripening. Duclaux' "casease"[1] was obtained by the author when he caused a precipitation of broth cultures of Tyrothrix tenuis, a bacterium belonging to the hay bacillus group. Babcock and Russell's "galactase" was found in the milk of cows, sheep, goats, pigs, horses half-breed buffaloes and in human milk. The authors consider galactase of importance in the ripening of cheddar cheese and cottage cheese and believe it to be a true milk enzym.

The presence of enzyms in milk has aroused some interest on the presumption that they enhance the digestibility of milk. Since enzyms are thermolabile substances the wisdom of pasteurizing milk has been questioned. This subject will be discussed later.

Some tests to determine whether milk has been heated or whether milk has been properly pasteurized have been devised based on the fact that heat affects and ultimately destroys enzyms. Storch's test is best known and is carried out as follows: 10 c.c. of a 3 per cent. solution of hydrogen peroxide are mixed with 20 c.c. of milk, and to the mixture 1 c.c. of a 2 per cent. solution of paraphenylin-diamin-chlorid is added. If the milk has been heated to 80°C. or above the reaction is negative; if below this temperature a blue color develops. Arnold's reaction is used for the same purpose. Guaiac resin is dissolved in alcohol or acetone to make a 5 or 10 per cent. solution and 0.5 c.c. added to 5 c.c. of milk. If the milk was heated to 72°C. for 15 minutes the reaction is negative, while a blue color appears in milk heated to a lower temperature.

These reactions, and similar ones that have been proposed, are of little use. In the first place milk is pasteurized in this country at a temperature too low to give a negative reaction, and furthermore, since pasteurization does not destroy all bacteria, those which remain grow and elaborate a fresh supply of enzyms.

THE GERMICIDAL ACTION OF FRESH COW'S MILK

Students of the bacteriology of milk made the curious observation that in fresh milk there was little or no growth of bacteria, and frequently even a decided decrease in numbers. Fokker advanced the theory that bactericidal substances were present in milk and that these were responsible for the restraining or destroying effect of fresh milk on bacteria. Ehrlich and Brieger showed that antitoxins and bactericidal substances are transmitted through milk, and later a number of authors believed they had proof of the existence of such immune bodies in milk. Stocking took a different view claiming that the reduction of bacteria in fresh milk was due to the death of tdose bacteria which found milk an unsuitable medium for growth Some light has been thrown on this subject by Rosenau and McCoy and Heinemann and Glenn. According to the former authors the reduction of bacteria in fresh milk is apparent only and due in large measure to the presence of agglutinins, while the latter authors hold that there are some bacteria which are actually destroyed, some merely restrained, and others not affected at all.

The facts seem to be these: In fresh milk there is a period during which bacteria do not multiply at the rate expected in a food substance which is eminently favorable for bacterial growth. This period varies in length of time according to the temperature at which the milk is held. At low temperature the phenomenon lasts for 12 to 24 hours; at room temperature for six to eight hours; and at body temperature for three to five hours. The phenomenon is observed in milk heated to 55°C., but is not so pronounced and above 68°C. there is no noticeable restraining influence upon bacteria.

The facts stated lead to the inference that immune bodies are present in milk, since the influence of incubation temperature and higher temperature is similar to the influence of temperature on immune bodies studied *in vitro*. However, the immune bodies are present in small quantity, owing to the enormous amount of milk produced by a cow in comparison to the blood supply, and because globulins are present in very small proportion. It is assumed that the antibodies are tied to the globulin fraction of blood protein.

The germicidal action of milk is not a practical issue since milk is commonly consumed at periods far beyond that of germicidal activity.

THE TRANSMISSION OF ANTIBODIES THROUGH MILK

Since it is known that some substances, as potassium iodid, sodium salicylate, bromids, mercury and arsenic salts, carbolic acid, aspirin, ether, chloroform, asafetida, can pass through the mammary gland and be recovered from the milk, it is possible that vegetable poisons may likewise do so. It is not probable, however, that this happens often, and if it does the poison in the milk is highly diluted.

Ehrlich has shown that antibodies can be transferred to the young through the milk of the mother. He immunized a pregnant mouse and when the young were born exchanged them for a litter of young mice from a normal mother. The immune bodies of the immunized mouse were transferred to the young mice from the normal mother.

Any antibodies in milk are highly diluted. They pass through the intestinal mucosa into the circulation. This was shown by Römer and Much who injected a pregnant cow with tetanus antitoxin. During the first days of life the young calf absorbed much more antitoxin through the intestinal mucosa than during the following days. When tetanus antitoxin was mixed with milk and fed from a bottle a much smaller amount was absorbed than when the milk was sucked directly from the mother. Antitoxins then, are readily absorbed by the young when they are derived from a mother who has been actively or passively immunized, but antitoxin is absorbed in small quantity only when mixed with artificial food. It has also been shown that more immune bodies pass into the system of the young when the lactation period of the mother is prolonged than when it is of short duration.

Besides antitoxins, agglutinins, bactericidal substances, hemolysins, opsonins and hypersensitiveness seem to be transmissible through milk.

Milk and the milk proteins can act as antigens. The corresponding antibodies are formed when milk proteins are injected into animals. As these may pass into the circulation by way of the intestinal mucosa of very young infants it is not surprising that precipitins form in the blood.

Adulteration of milk with that of another animal can be detected by means of the precipitin test even if the milk has been heated. If, for example, goat's milk were fraudulently mixed with cow's milk the deception could easily be proved by diluting the milk and mixing it with a serum from a rabbit immunized with cow's milk. The formation of a precipitate indicates the presence of cow's milk.

Antitoxins belong to the first order of immunity according to Ehrlich's theory. Agglutinins and precipitins belong to the second; and hemolysins and bactericidal substances to the third order. To prove the presence of antibodies of the third order two substances must be present, namely, amboceptor, the true antibody, and complement. It has been repeatedly observed that mother's milk did not seem to contain certain antibodies of the third order, while the blood of the offspring did. It is probable that the phenomenon is due to the often observed fact that complement is either wholly absent from milk or present in exceedingly small quantity. When milk containing amboceptor is absorbed by the young, the blood furnishes the necessary complement to give a positive reaction.

In milk derived from diseased cows complement is usually present in larger quantity than in the milk from normal cows.

THE BACTERIOLOGY OF MILK

It has been stated in another connection that milk in the udder contains bacteria, but that these are probably derived from without and are not of hematogenous origin. The possibility of bacteria passing from the blood through the mammary gland is not entirely excluded, but there is no positive evidence in favor of this assumption. The bacteria commonly found in the udder gain access through the folds of the teat duct. There is considerable evidence that under normal conditions milk is secreted from the mammary gland free from microörganisms. Experiments made by inoculating the blood of healthy cows with cultures of bacteria have failed to show their presence in the product and the fact that sometimes small quantities of sterile milk were obtained from the upper regions of the udder supports this view. At any rate, if the cow is normal the bacteria which gain access to the milk from without are of chief interest. These bacteria are derived from a variety of sources and unless great care is exercised in methods of handling milk enormous numbers of bacteria can be demonstrated in the final product.

After bacteria have entered the milk, and there is no market milk free from them, the milk must be cooled rapidly and kept cool in order to prevent their multiplication. There are some authorities of the opinion that lack of proper refrigeration after milking, and during transportation, is the most salient point to be guarded against, for it is known that many bacteria which gain access to milk find it a favorable medium and grow at an enormous rate after the initial "germicidal" period has expired.

Microörganisms are not evenly distributed in milk; samples taken from the same bottle or can, even after shaking and mixing, show different counts of bacteria, sometimes varying within wide limits. Bacteria occur in milk in clusters which are often difficult to break up. When milk stands, bacteria settle in part to the bottom and rise in part with the fat globules, so that cream usually contains many more bacteria per c.c. than the milk from which it was separated. This is true chiefly of gravity cream, since this process allows sufficient time for the growth of bacteria. Furthermore creaming of milk is often carried on in shallow pans with the result that the exposure of a large surface to the air increases the bacterial content.

Fresh separator cream is neither exposed to long standing, nor to the dust of the air, and if the separator receives the proper care the bacterial content of separator cream is far smaller than that of gravity cream. However, after cream has reached the market sufficient growth of bacteria may have taken place to greatly increase their number.

The bacteria found in milk and cream are usually harmless varieties, but milk has not infrequently been the carrier of infectious material. This fact makes it imperative to use all possible care to exclude pathogenic germs, and this is a difficult task. This subject will be discussed in a subsequent chapter.

The number of germs counted in market milk has been frequently only too high. In Washington, D.C., Rosenau counted 11,270,000 per c.c.; in New York City, Park reported finding 300,000 to 5,000,000; in Boston, Sedgwick and Batchelder found an average of 4,500,000; and in Chicago, Jordan and Heinemann counted 9000 to 18,000,000. At the present time the numbers will generally not reach such high figures, since pasteurization of milk is practised on a large scale with the consequent reduction of the germ content of milk.

It should not be overlooked that some of the germs in milk are beneficial. Buttermilk may contain hundreds of millions of bacteria per c.c. and still be entirely wholesome. The aroma of butter is largely due to microbial activity and the flavor and digestibility of cheese depend upon the presence of bacteria and molds. Scientific investigations have determined the exact types of microörganisms active in the ripening of dairy products and butter, butter milk and some cheeses are now manufactured by the use of pure cultures of bacteria and molds. The advantages of this method are these: (1) The milk can be heated and then inoculated, and pathogenic bacteria, if present, can be destroyed. (2) The quality of the product can be controlled.

When milk stands for some time it usually turns sour and forms a more or less firm coagulum, a change due to the action of microörganisms. This action does not cease with the coagulation of milk but continues until finally a turbid, foul-smelling liquid remains. This transformation of milk is a gradual one and occupies more or less time according to the temperature to which it is exposed and the types of microörganisms present. Although the decomposition of milk takes place gradually, it can be considered as occurring in five phases due to successive activity of certain groups of microörganisms. The first phase comprises the hours immediately after milking and may be termed the "incubation period." The changes during this period are not very marked, especially in clean milk. All bacteria that are able to utilize milk constituents grow slowly, if at all, during this period and their products neutralize each other in a measure. Some types are restrained by the early germicidal action of milk (page 541). The lactic acid bacteria grow from the start, but the acid content does not increase materially. This is partially due to the small number of lactic acid bacteria present; to the escape of carbon dioxide; to the fact that some of the acid combines with the casein; and to the production of alkali by some types of bacteria, this alkali combining with the acid formed. This incubation period rarely lasts longer than 12 hours. After this the second phase commences. Lactic acid bacteria chiefly of the Bacillus coli group, grow rapidly; the acid content increases; proteolytic bacteria are restrained by the acid formed; and some gas is formed, the amount varying with the number of bacteria present.

In the third phase the acid content is distinct; proteolytic bacteria are entirely restrained; streptococcus lacticus multiplies rapidly and the B. coli group grow more slowly, because they are less resistant to acid than Str. lacticus. The last mentioned organism, finally, produces enough acid to inhibit its own growth which ends when 0.9 to 1 per cent. acid has been formed.

During the fourth phase molds and yeasts multiply, as they prefer a decidedly acid medium. The yeasts consume the milksugar and the molds decompose the proteins with production of alkali. The alkali thus formed gradually neutralizes the acid and when this has disappeared conditions are ripe for the development of the spores of proteolytic bacteria. The result is a deep cleavage of proteins. The end products are mostly soluble and evil-smelling gases escape.

As intimated before, these phases are not necessarily distinct, but may overlap and the whole process may extend over a considerable period of time. The numbers of microörganisms, the types present and the temperature, all these factors are of paramount importance in influencing the character of the decomposition of milk.

There are chiefly five sources from which bacteria in milk are derived, namely: (1) Contamination from the udder; (2) contamination during milking operations; (3) contamination from utensils etc.; contamination during transportation; and (5) contamination in the hands of dealers and consumers.

Contamination from the Udder.—Contrary to earlier beliefs it is now conceded by leading authorities that bacteria are in the milk before it leaves the udder. The first streams of milk usually contain large numbers of bacteria; the middle milk a considerably smaller number; and the strippings according to some authorities contain very few bacteria, while according to others they contain more than does the middle milk.

The number of bacteria contained in the udder is variable, but usually small. Milk from different quadrants of the udder of a cow may contain different numbers.

Sometimes milk in the udder contains relatively large numbers of bacteria, even several million bacteria may be counted, but as a rule the number is very small, frequently below 100 per c.c. According to Harding and Wilson the germ content of milk in the udder averages somewhat less than 500 per c.c.

The type of bacteria found in the udder is chiefly the group of micrococci which constitutes about 75 per cent. of the germ content. Conditions for bacterial growth in the udder are not favorable and many types die when introduced into the udder. Streptococci are also frequently present, but usually in small proportion with some exceptions. B. coli has also been occasionally found in the udder. Esten and Mason found the following bacteria in milk obtained directly from the udder: Micrococcus lactis acidi, M. lactis albidus, M. lactis varians, M. lactis aureus, Bacillus subtilis. Harding and Wilson have described 71 groups of bacteria obtained from milk directly from the udder and 75 per cent. of these bacteria were micrococci.

Contamination during Milking Operations.—The young mammal obtains its milk supply by direct suction with the result that there is no contamination of the milk, excepting from the bacteria present in the udder and those derived from the skin of the teats. Milk artificially drawn for human consumption requires much handling and the possibilities for bacterial pollution are correspondingly increased. The chief sources of bacteria during milking operations are: (1) Dirt from the coat of the cow; (2) dust from the air; and (3) dirt from the hands and clothes of the milker.

During milking operations dirt and dust are shaken from the coat of the animal and these may find their way into the milk. The importance of this is emphasized by the fact that ordinarily cows are not curried, but dirt allowed to remain on the skin and dry. The picture of a cow with filthy flanks and legs is only too common. It is not surprising that investigators have found this neglect one of the most prolific sources of bacteria in milk. Part of this filth is soluble and remains in the milk; the insoluble portion may remain, or may be removed by strainers or centrifugal clarifiers. The number of bacteria contained in dust from the cow's coat is very great. Esten and Mason found 207,000,000 bacteria in 1 gm. of curry powder.

It is obvious that cleaning the udder and flanks of the cow before milking must reduce the germ content materially. In the best dairies this practice is universally observed and the skin is usually allowed to remain moist so that remaining dust particles adhere and do not drop into the milk.

The coat of the animal is a more serious source of bacterial pollution in winter than in summer, because during the winter months cows are confined largely to the stable where dirt may accumulate. Similarly, the morning's milk, drawn after the cows have spent the night in the stable, usually has a greater germ content than the evening's milk.

Cleanliness in the stable, periodic currying of the cows and cleaning the udder and flanks before milking will materially reduce pollution originating from the coat of the cow.

The air of the stable contains more or less dust and when this drops into the milk the germ content is increased. It is, therefore, a good policy to build stables with this fact in mind. Exposed rafters, ledges, sharp corners, etc. should be avoided. The floor should be of some absorbent material in order that it may be kept moist and dust kept down. The walls should be smooth and the ceiling tight.

Dust of the air, however, does not seem to be a very important source of pollution, although formerly it was considered so. Ruehle and Kulp, who have made a very exhaustive study of this subject, have come to the conclusion that the barn contributes about as much germ life to milk as the udder, that is to say, that in dusty barns of the ordinary type about 500 bacteria are added per c.c. of milk. It is of course desirable to eliminate this number as nearly as possible, but comparing this source of pollution with other sources it is relatively insignificant.

The milker may become a formidable source of bacterial pollution of milk. He should practice the utmost personal cleanliness and wear clothes that are as free from dust as possible. Investigations have shown that many bacteria may be added to the milk if the milker does not wash his hands before the milking operation. This is especially true when the milker, as is only too often true, wets his hands with milk before milking. "Wet" milking is a less laborious manipulation than "dry" milking, but is not sanitary and a prolific source of bacteria.

The milker is a menace not only because he may increase the bacterial content of milk, but because some of these contributed bacteria may be pathogenic. A germ carrier, one who carries typhoid bacilli, diphtheria bacilli, tubercle bacilli or the virus of scarlet fever, etc., can easily communicate the infection to milk and be the cause of disease and death. Dairies should be very careful in the selection of help and strict supervision of the health and habits of milkers will save much trouble.

The style of pail used for receiving the milk is a matter of the utmost importance. Wooden pails are entirely unsuitable, of course, as it is almost impossible to clean them thoroughly. But any pail of the old pattern with no protection at the top will admit many more bacteria than the style known as the small-top pail which is now becoming popular. This pail is covered except for an opening of 6 to 8 in. in diameter. By using such a pail dust and droppings from the animal are very largely excluded. There are several patterns of small-top pails in the market, some of which have sieves of cotton or brass, others none. In a cleanly dairy no sieve is needed and as a matter of fact is a disadvantage, as it requires extra cleaning and the dirt particles remaining on the sieve are broken up by succeeding streams of milk until bacteria can pass through and reach the milk. In an ordinary dairy the sieve may be advantageous.

According to numerous investigations the germ content of milk can be reduced to one-seventh and even less by using a small-top pail in place of the old-fashioned open pail.

During recent years milking has been performed by machines in many dairies. It was hoped that by the use of machines some of the important sources of bacterial pollution could be eliminated and in a measure at least this hope has been realized.

Milking machines have been used for a considerable length of time, although the original stimulus was the idea of labor saving rather than the production of sanitary milk. Modern milking machines are operated by intermittent suction and the fact that the milk flowed directly into a pail from the udder with apparently no contact with the air gave rise to the expectation that the germ content would be materially reduced. In the early history of milking machines this expectation was not realized. Indeed, the milk obtained by machine showed 10 to 20 times the number of bacteria contained in milk obtained by hand milking from the same animals. But scientific research has largely overcome the difficulties so that at the present time milking machines are used with success.

The first difficulty was experienced in cleaning the parts of the machines. The rubber hose used retained bacteria tenaciously, but later experiments showed that immersion in brine, after cleaning, sterilized it, providing, however, that no air was left in the hose, so that the brine came in intimate contact with all parts.

The construction of the teat cups offered another difficulty. The size of the cow's teats varies greatly and even teats of the same udder are not always of the same size. Therefore, the cups would frequently be so loose as to allow air to be sucked in, or they would fall to the floor and become contaminated. In modern construction a rubber veil forms part of the teat cup and as this veil is always tight against the teat it overcomes all difficulties.

The suction in modern milking machines is intermittent and a periodic relief is therefore necessary. Whenever this relief opens, dusty stable air is admitted to the system. This objection has now been overcome by inserting relief cotton filters. The air that is sucked in is then filtered through this cotton and the dust and bacteria removed.

A further source of contamination was found in the fact that condensation water would enter the milk during the intervals when suction stopped. By inserting efficient traps this difficulty was successfully overcome.

It was once thought that cows would not accustom themselves to machine milking and would so withhold milk as to reduce the yield and shorten the lactation period. Careful observations, however, seem to indicate that cows may readily become accustomed to milking machines and that milk secretion is not decreased by them.

Contamination from Utensils.—It is quite obvious that poorly constructed and poorly cared-for utensils constitute a most prolific source of bacteria. In fact some authors consider this source the most important one. Utensils should be free from corners, dents and imperfections which permit small amounts of milk to remain and serve as food for bacteria. Even well-constructed utensils soon become a menace if they are handled roughly. Strainers, all sorts of machinery and the bottles require care to prevent the multiplication of bacteria.

Mere washing is not sufficient, utensils must be treated with boiling water or better with high pressure steam. It is difficult under ordinary dairy conditions to sterilize utensils in the scientific sense, but they can be treated in a manner to destroy nearly all germ life. Investigators have found 10 to 100 times as many bacteria in milk drawn into unsterilized pails as into sterilized pails, other conditions being as nearly equal as possible.

All utensils should be first washed in cold water to which some alkali has been added, then washed in clean water and finally steamed. Some dairies have an appliance which holds the cans so that steam under pressure can be introduced. When the producer is unable on account of lack of facilities to sterilize his cans the creamery should do so in order that the cans be returned in sterile condition. It is a good practice to tie a piece of cloth over the top of sterilized utensils and allow this cloth to remain in place until the utensils are to be used, as this keeps dust and flies out of the cans.

Special attention should be given to milk bottles, not only as they may be the source of many bacteria, but as they may have been in homes where infectious diseases exist and thus become the means of distributing pathogenic organisms. Milk in bottles should be protected against attacks from domestic animals. They are frequently left in the open before they are taken into the house and may then be a temptation to cats and dogs. Every household should be provided with a box, preferably one insulated, to receive the milk when it is delivered early in the morning.

The Influence of Temperature on the Bacterial Content of Milk.—The chief factor during transportation of milk is proper refrigeration. If the temperature is not sufficiently low there will be an enormous multiplication of bacteria as the following figures determined by Hunziker show:

Original number of bacteria	5,000 per c.c.
After 24 hours at 42°F	2,400 per c.c.
After 24 hours at 50°F	7,000 per c.c.
After 24 hours at 65°F	280,000 per c.c.
After 24 hours at 95°F	12,500,000 per c.c.

Refrigeration during hot weather is more difficult, of course, than during cool weather. Jordan and Heineman found in Chicago milk that during the month of April the average number of bacteria per c.c. was 9,361,000; during May which was cool a month it was 10,071,000; and during June 18,924,000.

These figures clearly show the importance of keeping milk cool up to the time of consumption. The temperature should not be above 50°F., and it is more desirable to have it below this figure.

Large communities depend upon a milk supply that may have to travel a long distance. Unfortunately some railroads are not equipped with suitable refrigerator cars and rarely run milk trains on a sufficiently fast schedule.

Contamination of Milk in the Hands of Dealers and Consumers.—Milk delivery wagons should be provided with cooling facilities. Milk is sold chiefly in bottles so that cooling in wagons is facilitated. Dipping milk from cans is not as common as formerly, and should be entirely abolished, since it is not only difficult to keep the milk cool, but the frequent handling exposes it to bacterial pollution.

In the home milk should be placed on ice in warm weather and kept there, to be removed only for consumption. Ordinary refrigerators are not efficient in keeping milk cool enough.

The bottles should always be covered so as to exclude dust and flies. There are several inexpensive devises in the market for covering milk bottles after the usual pulp cap has been removed. The pulp cap should be wiped with a clean cloth before the bottle is opened. Both cold and hot weather expand milk and a few drops are liable to ooze out and remain on the cap. It is evident that these drops may become polluted, hence no cap should be removed until it has been cleaned. After the milk has been emptied the bottle should be washed, first in cold water, then in hot soapsuds and then rinsed with clean hot water.

In order to obtain a clean milk there have been applied a number of schemes, most of which are calculated to remove dirt and bacteria rather than prevent their entrance. Filtration of milk through sand has been practiced in Denmark. Filtration through flannel or similar material has been tried in this country; centrifugal clarifiers are used extensively to throw out the dirt; and in many dairies the milk is strained two or three times before it is bottled. There can be no objection to straining milk once, because occasionally the presence of some dirt particles cannot be avoided, but straining more than once increases the bacterial content and exposes the milk to renewed contamination.

The most reasonable method for producing clean milk is the use of proper precautions in its production so that excessive quantities of dirt do not enter. All methods of filtration and straining cannot make the product equal to one obtained under the best sanitary conditions, since a considerable quantity of dirt is soluble and cannot be removed by any subsequent treatment.

The dirt that gains access to milk during barn operations is chiefly cow manure, and while there is small danger of pathogenic bacteria entering from this source, clean milk is desirable and can be produced without great expense. All food is expected to be clean and there is no reason why milk should be an exception. If milk were a transparent fluid the millions of bacteria present in some market milk would render it turbid and a source of alarm to the consumer.

Since milk is an excellent food for microörganisms the kinds of bacteria found in milk have been studied with much interest. The variety of types found in milk is so great that the term "milk bacteria" is frequently encountered. However, it is necessary to consider this term a misnomer, as milk is believed to be secreted by the mammary glands devoid of germ life. Therefore, inherent "milk bacteria" cannot exist.

It is a fact that certain types of bacteria in milk are encountered with astonishing regularity. This is exemplified in the decomposition of milk which occurs with few exceptions in a certain manner. This fact is easily explained, however, since during the production of milk a limited number of types enter and the sources of these types are fairly uniform. Furthermore, there are certain types of bacteria which grow in milk with great rapidity, soon outstripping other bacteria so that the results of necessity must be quite uniform.

It is true that some bacteria after a lengthy sojourn in milk so alter some of their properties as to accommodate themselves to the environment. But this would and does happen in any sort of medium and does not justify division into distinct types.

There is in the literature an overwhelming nomenclature given to types of bacteria which are discovered under different conditions and, therefore, have assumed somewhat different properties. For example: The lactic acid bacterium "par excellence," Streptococcus lacticus, has been given more than 50 names by different authors. And furthermore, similar organisms have been described by pathologists under various new names and much confusion has resulted. It should be borne in mind that bacteria are very variable, probably more so than any other group of organisms, and modern bacteriologists are loath to apply the term "species" to bacteria.

The chief groups of microörganisms occurring in milk are: (1) Lactic acid bacteria; (2) spore-bearing bacteria; (3) bacteria causing abnormal conditions in milk; and (4) molds, yeast and torulæ.

The lactic acid bacteria have received more attention by investigators than any other group present in milk. They have been studied, classified and grouped by many. Perhaps the most complete classification is that of Löhnis which arranges lactic acid bacteria in four groups. One of these groups is the Bacillus coli group. B. coli has been found to be always present in market milk and even in the majority of samples of certified milk. It attacks milksugar with the formation of acids, including lactic acid and several volatile acids; it forms gas and attacks the proteins slightly in the presence of carbohydrates, more profusely in the absence of carbohydrates.

Bacillus aerogenes is also a constant inhabitant of milk and belongs to the B. coli group. It attacks carbohydrates vigorously with acid and gas formation, but as a rule does not decompose proteins.

The lactic acid organism described by Hüppe and known as Bacillus acidi lactici belongs to the B. coli group and is considered a variety of B. aerogenes.

The second important group of lactic acid bacteria is the streptococcus group. Str. lacticus grows preferably under anerobic conditions and is found most abundantly in the deep layers of souring milk. It produces more acid than B. aerogenes and the acid is almost pure lactic acid. No gas is formed. The organism is known in the literature under different names, the commonest one being B. lactis acidi, Leichmann. Thit organism is still sometimes confused with B. acidi lactici, Hüppe. It is the lactic acid bacterium par excllence. Strains of Str. lacticus produce agreeable aroma in butter and are the agents in the formation of ordinary buttermilk.

The so-called "spontaneous," "natural," or "normal" souring or loppering of milk is due to the action of the two groups of organisms mentioned. The B. coli group initiates the formation of acid and the streptococcus group completes it, although the latter group produces acid simultaneously with the B. coli group.

The coagulum in sour milk varies according to the predominance of either group. When the B. coli group predominates, gas is abundant and is indicated by bubbles in the curd, and the aroma is usually not agreeable. The curd contracts rapidly and whey is liberated in considerable quantity. The curd formed by Str. lacticus is smooth; there are no gas bubbles; whey is separated in small quantity; and the taste is agreeably sour.

The bacteria of the B. coli group are derived almost exclusively from fecal matter, while the streptococcus group, although also largely derived from fecal matter, occurs in other places. In milk which is heavily polluted with manure large numbers of B. coli are found and the results of the souring process are not as desirable as those obtained from clean milk in which B. coli is present in relatively small numbers.

The temperature at which the acid fermentation takes place has of course considerable influence on the result. At 30°C. and above the B. coli group grows rapidly creating their peculiar effects, while at moderate temperature, from 20 to 25°C. the streptococcus group soon outstrips the B. coli group. Milk which sours at a moderate temperature has, therefore, a more agreeable flavor than that which sours at higher temperature, other conditions being equal.

B. coli and Str. lacticus utilize chiefly the milksugar for food, but while the former may also split proteins, Str. lacticus attacks proteins very feebly. However, B. coli splits proteins in appreciable quantity only when carbohydrates are absent, with the result that the split products are less disagreeable when carbohydrates are present than otherwise.

It is obvious that the best quality of buttermilk is obtained when the milk is sufficiently heated to destroy most of the bacteria and then inoculated with a pure culture of Str. lacticus. This is now commonly done, not only in the manufacture of buttermilk, but also in the making of butter. By inoculating pasteurized cream with cultures of Str. lacticus the "ripening" or souring of cream proceeds normally and the product is of uniformly high quality. The most desirable curd for cheesemaking is also produced by Str. lacticus, while B. coli, if present, in large numbers, is liable to produce off-flavors.

The third group of lactic acid bacteria has received considerable attention during recent years and is spoken of as the B. bulgaricus group or the lacto-bacilli. These are large bacilli and produce considerably more acid than either of the two previously mentioned groups. There are two distinct classes of lacto-bacilli. One class forms an average of 1 to 1.8 per cent. acid consisting almost entirely of levorotatory lactic acid, while the other class produces more acid, sometimes over 3 per cent. and even as high as 4 per cent. The acid consists chiefly of inactive lactic acid. The first class of lacto-bacilli stains in granules with alkaline methylene blue, or Neisser's stain, and the bacilli sometimes have been termed "Körnchen" or "granule" bacilli. The bacilli of the second class stain solidly. Both classes are Gram-positive in young cultures, but older cells are Gram-negative. A Gram stain prepared from cultures 24 or more hours old and counterstained with fuchsin frequently shows a beautiful picture of filaments in which blue and red cells occur, the latter being the Gram-negative forms.

The curd formed by lacto-bacilli in milk is characteristic. The high-acid forming type usually forms a smooth, semi-fluid curd and whey is either absent or present in extremely small quantity. The low-acid type forms a curd of less smoothness, but still smoother than that formed by the B. coli group. More whey appears when the low-acid type coagulates milk than when the high-acid type does, but the quantity is never large.

The lacto-bacilli have long been overlooked, owing to the fact that they do not grow, or at least grow very sparingly, on ordinary laboratory media. Carbohydrates are essential to their growth and milk is by far the most suitable culture medium. They are also sensitive to temperature conditions. At ordinary temperature most strains do not grow at all; at 30°C. they thrive sparingly; and the optimum temperature of many strains lies at about 44 to 45°C.

Lacto-bacilli were first isolated from some fermented milks, chiefly Yoghurt of the Bulgarians and Leben of the Egyptians. Later they were found under many natural conditions and are almost universally present in milk, even in certified milk. When milk is kept at a temperature of 37°C. or above, it becomes intensely sour due to the presence of lacto-bacilli. The high acidity prevents growth of other bacteria so that such milk does not putrefy like ordinary loppered milk.

Lacto-bacilli have recently been found to play an important role in cheese ripening. Evans, Hastings and Hart found them in large numbers in ripe cheddar cheese, and Eldredge and Rogers reported finding them in ripe Emmenthaler cheese. Esten and Mason isolated them from Camembert and Roquefort cheese.

Heinemann and Hefferan found lacto-bacilli in many substances, such as silage, bran, sour-krout, pickles and in the feces of man, horse and cow. The authors further showed their presence in both normal gastric juice and in gastric juice obtained from patients suffering from carcinoma of the stomach. The so-called Boas-Oppler bacillus was proved to be a lacto-bacillus by Heinemann and Ecker. Dotterer and Breed found that pasteurized whey from cheese factories contained lacto-bacilli almost in pure culture.

Lacto-bacilli have been described under a variety of names, as for example: B. bulgaricus, B. lebenis, B. panis fermentati, B. casei, Boas-Oppler bacillus, B. of Tissier etc.

Lacto-bacilli, as far as present knowledge goes, are non-pathogenic. They have been recommended by many authors, chiefly Metchnikoff, for therapeutic purposes. This author considers ingestion of the bacilli, either in cultures, tablets or in sour milk, as a means of deferring premature senility. The theory advanced is based on the high acid-producing properties, inasmuch as it is assumed that the acid formed in the intestinal tract prevents putrefaction. Cultures of lacto-bacilli have also been used in the treatment of pathologic conditions accompanied by pus formation and in the treatment of intestinal disturbances of infants. This subject needs closer investigation before definite conclusions can be reached. Some benefits undoubtedly have been derived from the consumption of Bulgarian milk, but whether they were caused by the prevention of putrefaction or simply by the introduction of an easily digestible food is an open question.

The fourth class of lactic acid bacteria consists of micrococci, closely related to the staphylococci. They produce acid and break down proteins and play a role in the ripening of cheese. Otherwise they are not of great importance.

Besides lactic acid bacteria other types of microörganisms are commonly found in milk. Members of the hay bacillus (B. subtilis) group are nearly always present. These organisms digest proteins and some strains produce a coagulative enzym which is the cause of a phenomenon known as "sweet curdling" of milk.

Some anaerobic spore-bearing bacteria, such as B. butyricus, B. putrificus, B. welchii, are commonly present in milk. Ordinarily these do not grow in milk, but sometimes conditions are such that the milk is decomposed by these organisms. Usually a clot is formed which is broken up by gas bubbles and much whey is separated.

Chromogenic bacteria are not infrequently present in milk and, although under usual conditions they are restrained from appreciable growth by the acid formed by lactic acid bacteria, they sometimes gain the upper hand and impart their pigment to the milk. Blue milk is sometimes encountered and is produced by B. cyanogenes or Bact. syncyaneum. Red milk is produced by B. prodigiosus or B. erythrogenea; the former usually coloring the cream only, while the latter colors the whole volume of milk. A sarcina forming a red pigment has also been found to be the cause of red milk. Yellow milk formed by Sarcina lutea is rarely observed and the pigment does not stay in suspension, but settles rapidly. Bact. synxanthum forms a yellow pigment which diffuses throughout the milk.

Pigment-forming bacteria are harmless and objectionable only because the milk assumes an unnatural color.

Not infrequently milk is observed to become slimy or stringy. When poured slowly from one vessel to another threads several feet in length may form. The commonest organism causing slime formation in milk is a bacillus occurring widely in standing water. The bacillus does not grow well at body temperature, but requires relatively low temperature. Milk may therefore become slimy, when kept in an ordinary home refrigerator. Str. lacticus sometimes and also some strains of B. bulgaricus acquire the property of forming slime in milk.

A bitter taste sometimes develops in milk caused by several organisms. Proteolytic bacteria dissolve the proteins with production of peptones which have a bitter taste. These organisms are derived from cattle foods. Micrococcus lactis amari of Conn, and Torula amara of Harrison, may be the cause of bitter milk. The organisms grow when lactic acid bacteria are scarce.

B. saponaceus causes a soapy taste in milk and causes it to foam as though there were soap in it.

Other off-flavors are: A fruit-like odor, especially a flavor reminding one of strawberries is produced by some strains of B. coli; a butter flavor caused by Conn's culture 41; an unpleasant taste and odor produced by Marshall and Farrand's Bact. fulvum; and a rancid odor caused by Löhnis' Bact. Kirchneri.

All the organisms mentioned causing abnormal conditions in milk are harmless, but change it in a manner to render it distaseful. It is, therefore, important that the dairyman get rid of them. It is sometimes not easy to locate the exact source of trouble. The cow, the stable, utensils or machinery may be responsible. If the cow is the cause she must be removed from the herd. If some condition in the stable is responsible it should be remedied, and if utensils or machinery are contaminated milk of lime offers a good disinfectant.

Among molds found in milk Oïdium lactis is the most prominent. It forms a felt-like layer on curdled milk, digests proteins and imparts a peculiar odor and taste, reminding one of limburger cheese. Penicillium, mucor, and other molds also commonly occur in milk. Torulæ and yeasts are so widely distributed in the air that milk is rarely free from them. Yeasts attack the milksugar with production of gas and esters which impart characteristic flavors to fermented milks. Some types of Torula ferment lactose with violent gas formation.

FERMENTED MILKS

Fermented milks are the result of microbial action and have been used by man for many ages. As a rule, several types of microörganisms act simultaneously; one type produces acid; another acid and carbon dioxid gas; a third flavor; and in some fermented milk the protein is partly broken down. Fermented milks change the character of milk in a manner to render it more stable and consequently its food value is largely preserved.

Fermented milks are either the result of acid fermentation or of acid and alcoholic fermentations combined. Buttermilk is formed by acid fermentation of Str. lacticus. Some of the milksugar is broken down and the casein is precipitated. Buttermilk is in reality the sour skim milk left in the churn after butter making, but with the introduction of cream separators the yield of buttermilk has become too small to fill the demand. Separator milk is now inoculated with cultures of lactic acid bacteria of the Str. lacticus type and churned after it has ripened. This is the buttermilk that is now commonly sold. Churn buttermilk may not contain the plasma solids in as large proportion as the buttermilk made from separator milk, because sour cream is frequently diluted with water by butter makers. The food value of artificial buttermilk, therefore, may be greater than that of churn buttermilk.

Tätté Melk, Tättemjölk or Kjernemelk is a thick, slimy product of fermentation used in Scandinavian countries. A slime-forming streptococcus and a yeast are the chief agents, although Oïdium lactis is also present. This milk food is prepared by placing a few leaves of Pinguicula vulgaris or a variety of Drosera in milk. It is also produced by inoculating sweet milk with some previously ripened milk, or by using pieces of linen that have been dipped in ripe milk.

Yoghurt in Bulgaria, Leben or Leben-Raïb in Egypt, Skorup in Serbia and Montenegro, Gioddu in Sardinia, Mazzorado in Sicily and Dadhi in India are fermented milks of similar properties. In all of these milks a lacto-bacillus is the acid producing organism and a yeast furnishes aroma. They are thick milks, frequently eaten with spoons and are incubated at temperatures ranging from 45 to 50°C., Yoghurt is prepared from milk after it has been reduced to one-half or one-third its volume by evaporation. The finished product, therefore, contains the milk solids in highly concentrated form.

The milk used for these fermented products is not always treated in the same manner. Yoghurt and Lebem are made from buffalo's, goat's or cow's milk; Skorup is made of cream separated from boiled milk; Urgoutnik, used in the Balkan mountains, is made from sheep's milk. The final products differ from each other according to the composition of the milk from which they are made. Sheep's milk is richer in fat and sugar than cow's milk and the fermentation, therefore, is greater and the aroma assumes the character of the original milk.

Mazun is another fermented milk rich in lacto-bacilli, but yeasts and a streptococcus are also present. A spore-bearing bacillus digests the casein to some extent so that the final product assumes a cheesy odor.

The fermented milks described are incubated at relatively high temperature and consequently alcoholic fermentation is held in check in spite of the fact that yeasts are abundant. Kefir and Koumiss are incubated at a temperature of 14 to 18°C. and alcoholic fermentation is quite distinct. Rogers suggests that a similar beverage can be prepared by inoculating buttermilk with yeasts capable of producing an alcoholic fermentation which is favored by adding some cane sugar.

Kefir or "best beverage" is used by inhabitants of the Caucasus mountains and is made from sheep's, goat's or cow's milk. Alcohol, carbon dioxid, glycerin, lactic acid, succinic acid, acetic and butyric acids are among its constituents. The proteins are slightly broken down. The ferment consists of grains of the size of millet seeds or larger and is considered by the natives a divine gift. The grains can be preserved for a long time and before using are soaked in warm milk or water. They swell, rise and are skimmed off. They are then ready to start the fermentation in milk. The inoculated milk is filled in skin bags and these are hung near a door, where they are agitated by passers-by. When the kefir is ripe a small portion is preserved for future inoculations. Kefir contains about 0.75 per cent. alcohol.

Koumiss or "milkwine" is used by the nomadic tribes inhabiting South Russia and Asia. It is made from boiled mare's milk and the fermentation is initiated by adding a small amount of ripe Koumiss to sweet milk. The Kalmucks prepare a brandy-like beverage from Koumiss by distillation. This is known as "Araka," "Rack," "Racky," or "Ojran" and contains 7 to 8 per cent. alcohol. Ripe Koumiss contains 3 per cent. alcohol and is effervescent.

CERTIFIED MILK

The late Henry L. Coit of Newark, N. J., conceived the idea of producing a milk under such precautions as to practically insure its cleanliness, uniformity of composition and low bacterial content. A special feature of the plan made it improbable that pathogenic bacteria could gain access. This milk was called certified milk and was intended to serve as food for infants and invalids. According to Dr. Coit's plan a medical society should appoint a commission of six or more members who would give their services free of charge and who would supervise the production. The commission should engage an expert chemist and bacteriologist to test the milk at regular intervals; send a physician to the dairy once a month to examine the employees; and have the cows examined and tested for tuberculosis by a veterinarian.

Certified milk requires exacting conditions. It must be produced in barns whose structure facilitates the exercise of cleanliness; the cows furnishing the milk must be kept clean by currying; their flanks and udders must be clipped and washed with a damp cloth immediately before milking; food must not be given these cows for at least one hour before milking; and the floors of the barn must be moistened before milking. The milkers must wear white suits and caps which are never worn for more than one day's milking; they must wash their hands each time before milking a cow; and they must never moisten their hands for milking. In addition the small-top pail is the only milking pail used and all vessels are sterilized with live steam. The milk is cooled as rapidly as possible after milking. Manure is removed daily from the stables and when not spread in the field, is carried to a pit at least 100 ft. removed from the stable. No live stock, excepting the cows, is permitted within 100 ft. of the barn, and the dairyman is obliged to carry out many more similar conditions.

All cows must be tested with tuberculin semi-annually and reactors immediately removed from the healthy herd. Newly acquired animals are placed in a quarantine stable and not used for milking before the tuberculin test has proved negative and veterinary examination has shown them to be free from disease. Food is carefully selected and no water used unless pure and wholesome.

The stables are located on elevated ground to facilitate drainage and must be constructed with tight ceilings. Food is carried into the stables from without and not, as is commonly done, lowered through apertures in the ceiling. The floors are of smooth concrete and the walls smooth. An efficient system of ventilation is installed in the barns and both windows and doors screened. The gutters and mangers have round corners to facilitate cleaning.

The experts employed by the commission procure milk samples from the open market and test the milk for chemical composition and bacterial content. The amount of fat demanded by different commissions is not the same, but the majority demand 4 per cent., allowing ¼ of 1 per cent. either way. The bacterial content should never be higher than 10,000 per c.c.

The owner or superintendent of a certified milk dairy is required to report promptly the appearance of any disease among the personel. This is an important condition and is calculated, of course, to prevent infectious material from entering the milk.

All expenses incurred in the supervision of certified milk production are borne by the producer. A formal contract is signed by him and failure to comply with all conditions may lead to the withdrawal of the patronage of the commission. In return for faithful compliance with all demands the commission authorizes the producer to place a certificate on each bottle of milk, and hence, the product is called certified milk. This certificate is virtually a guarantee of the commission that the milk is wholesome; that it contains the requisite food elements; and that it is free from foreign substances and pathogenic bacteria. It stands to reason that certified milk is sold at a higher price than ordinary milk.

The introduction of certified milk on the market has been of much benefit, not only because a reliable milk is available but because of the moral effect on milk supplies in general. The production of certified milk has shown that milk of the best quality can be produced and that its wholesomeness can be safeguarded. It has also been shown that expensive equipment, involving large pecuniary investment, is not essential to the production of clean, wholesome milk, but that methods employed are of paramount importance. Clean, wholesome milk cannot be produced by expensive equipment and slovenly methods, but can be produced with fair equipment and unimpeachable methods.

However, the certified milk producer has difficulties to contend with which may appear insurmountable. The germ carrier is only too common and he is frequently difficult to find. A germ carrier may be among the milkers and no one aware of this fact. Typhoid carriers, for example, discharge typhoid bacilli intermittently and repeated bacteriological examinations may fail to locate them.

Furthermore, the help question is frequently a very serious one. Milkers may have to be replaced and it hardly seems feasible to compel a dairyman not to allow a new man to milk cows before he has been subjected to a bacteriological examination.

A medical society milk commission must be constantly on the alert if the product is to comply with the demands; it must be painstaking; honest and interested in its work.

Unfortunately there is no general legal protection for certified milk. A few states, New York, New Jersey, Kentucky, Massachusetts and New Hampshire, have legislative protection for certified milk, but in the majority of states there is none. The only safeguard a milk commission has is to print on the certificate "certified by the ...... Medical Society Milk Commission."

From a commercial point of view the production of certified milk is not an unqualified success. Some producers consider themselves philanthropists, since their profit is so small, and unless the demand for certified milk increases considerably their stand is justifiable. The production of certified milk has been fostered by two associations. One is the Association of Medical Milk Commissions founded in 1907. This association attempts to regulate the requirements for certified milk and has published its demands for composition of the milk in the form of a score card. This card serves as a guide for commissions in the control of certified milk production. The second association, founded in 1908, is the Certified Milk Producers Association. Both associations work together in large measure with the view to increasing the output of certified milk and making it a success generally.

STERILIZATION AND PASTEURIZATION OF MILK

Soxhlet was the first to apply heat to milk in order to prolong its period of usefulness. In 1866 he had special bottles made which were to be filled with milk and after they were closed tightly were to be heated in boiling water. This process destroys the majority of microörganisms, but allows some spores to live. It is, therefore, not a process of sterilization, as Soxhlet thought.

Soxhlet's method was widely used and with considerable success. Milk remained sweet for a longer period than raw milk and the "sterilized" milk was used for infant feeding. In this country in 1889 Jacobi recommended the use of milk treated by Soxhlet's method.

However, some objections to this method were raised, chiefly because of the cooked taste which develops in milk heated to the boiling point. It was held, furthermore, that boiled milk was more difficult to digest than raw milk. It is true that at the boiling point milk constituents undergo considerable change in composition and due to these objections the process of pasteurization was introduced. Pasteurization is the process originally applied by Pasteur to the preservation of wine and beer. It is, therefore primarily, a process designed to preserve beverages, but in later times it has been applied, especially to milk, for a more important reason, to destroy pathogenic bacteria.

There are three methods of pasteurization in vogue, namely: (1) the flash process; (2) the holding process; and (3) the process of pasteurizing in bottles.

The flash process, also known as the continuous process, consists in heating milk to a relatively high temperature for a brief time. It was first practiced in Denmark on a large scale where it was applied chiefly to the treatment of skim milk from creameries, and whey from cheese factories. These by-products have been known to spread tuberculosis and other diseases and in Denmark the reduction of tuberculosis among cattle is due largely to the pasteurization of creamery and cheese factory by-products. The milk was heated for a short time to 85°C. (185°F.). At this temperature most bacteria are destroyed, but some, especially spores, survive.

Heating milk to this temperature influences the cream line as has been explained in a previous chapter. The volume of cream seems to be less than in the raw milk and the process of creaming is slower. There is, of course, no actual loss of fat. The phenomenon is due to the breaking up of fat globules. Consumers objected to the fancied loss of cream, and dairies began to lower the temperature of pasteurization and now the temperature most commonly applied for flash pasteurization is 160°F. (71.1°C.) This temperature is not entirely satisfactory, because, although the great majority of bacteria are destroyed, some of the survivors may be pathogenic.

In order to secure the destruction of pathogenic bacteria the holding or held process of pasteurization was introduced. By this process milk is held at a much lower temperature than in the flash process, but for a considerably longer period. The temperaure and the period of holding necessary to accomplish the purpose is based on the thermal death-point of the tubercle bacillus. This organism is more resistant to heat than other pathogenic bacteria occurring in milk, and therefore, any temperature that will surely destroy the tubercle bacillus will render milk safe from infectiousness.

The thermal death-point of the tubercle bacillus has been the subject of considerable controversy. Earlier investigators thought that it required boiling of milk or water to destroy this organism, but Theobald Smith showed that it was destroyed in milk by an exposure to 60°C. for 20 minutes, unless a film was allowed to form on the milk. This film protects the bacillus against heat so that a higher degree is necessary to destroy it. Smith's findings have been confirmed by Russell and Hastings, Rosenau and others. During pasteurization the milk is agitated to a sufficient degree to prevent film formation.

Under laboratory conditions 20 minutes heating at 60°C. is sufficient to render pathogenic bacteria in milk harmless, but this does not necessarily apply to pasteurization under commercial conditions. When milk is heated in large tanks, the temperature is not evenly distributed and some parts of the milk are not held at the right temperature for a sufficient length of time. Shorer and Rosenau, who have studied this problem, conclude that heating milk to 145°F. for 30 minutes assures a sufficient margin of safety.

A further difficulty under commercial conditions lies in the foam which forms on the surface of the milk. This foam protects the bacteria against heat so that some survive. The milk should, therefore, not be stirred with violence, but in a manner, which will avoid foam formation as much as possible.

The third method of pasteurization consists of heating the milk after it has been filled into bottles. After filling the bottles they are immersed in water and then gradually heated to pasteurization temperature, or warm water is sprayed on the bottles until the proper temperature has been reached.

At first glance pasteurization in the final package is the ideal method, because recontamination after pasteurization seems impossible. But there are some objections. Pasteurization in a large vessel removes bad odors, but in a closed bottle these odors are retained or even intensified. Only clean milk can be pasteurized in bottles to avoid this objection. A second difficulty lies in the fact that heat expands milk and the bottles must be large enough to leave an air space so as to prevent overflow of the milk when it expands. Finally, present costly machinery must be discarded and replaced by machinery of a different type, if this kind of pasteurization is to be adopted.

A way of combining the holding process with the last mentioned method has been devised by Ayers and Johnson. Milk is heated, held in a large vat and then filled while hot into hot bottles. Thus some of the objections are successfully overcome.

It is usually considered imperative that pasteurized milk must be cooled rapidly to prevent surviving bacteria from multiplying. Ayers and Johnson after studying this question state that there is no appreciable growth if the cooling process requires up to five hours. The surviving bacteria, it seems, are affected by the heat so that some time has to elapse before they are able to grow normally.

Pasteurization of milk is not universally recognized as an efficient safeguard against infection. It is frequently advanced that lactic acid bacteria are destroyed and that putrefactive organisms survive with the result that they decompose the milk and produce toxic substances. Ayers and Johnson, however, have conclusively shown that milk pasteurized by the holding process, or milk pasteurized at temperatures up to 160°F. for 30 minutes, contains lactic acid bacteria in greater proportion than does raw milk.

It is believed by some that pasteurization so changes the composition of milk as to render it less digestible. The investigations of Rupp have disproved this objection. This author found that the assumed chemical changes in milk pasteurized at 145°F. for 30 minutes cannot be detected by chemical means.

The producer who is ignorant of the true purpose of pasteurization may argue that he need take no precautions against uncleanliness, believing that pasteurization removes dirt. Careless production may thus be encouraged, but this may be overcome by efficient inspection of dairies. There is no reason why inspection should be relaxed because the milk is to be pasteurized.

Some objectors claim that enzyms in milk are destroyed by heat. At 145°F. most enzyms are not destroyed or even materially affected. Furthermore it has not been shown that the enzyms in cow's milk are of benefit to human beings.

It has been further objected that vitamins are destroyed by pasteurization. A great deal of experimental work has been done in order to prove or disprove this contention. The results, as far as modern knowledge warrants, may be summed up as follows: In milk pasteurized by the held process the fat-soluble vitamin *A* and the water-soluble *B* are not affected. The water-soluble vitamin *C* is destroyed, but since this vitamin is present in raw milk in insufficient quantity some addition to the diet becomes necessary in order to correct this deficiency. Orange juice, tomato juice, prune juice and similar antiscorbutic vegetable juices are required for a diet of raw milk as well as of pasteurized milk.

The objection that pasteurization is not always carried out conscientiously cannot constitute a valid one. The principle of pasteurization is correct and dishonesty or carelessness on the part of those who carry out the process cannot vitiate it. Health departments, to which is intrusted the inspection and control of food, must also inspect and control pasteurization plants in order to insure efficiency of the process.

The cost of pasteurization is a negligible factor. According to Bowen it costs but 0.313 cents per gallon of milk.

Home pasteurization is recommended by many and when carried out properly insures safe milk. It is not an easy matter, however, to impress the housewife with the importance of proper temperature. If, for example, the thermometer is not placed in the bottle containing the milk, pasteurization may be a failure. Intelligent use of the thermometer is absolutely necessary. In the home the safest control of unclean milk is to bring it to the boiling point.

Besides pasteurization there have been attempts at safeguarding milk by other means, although none have gained much popularity. The addition of hydrogen peroxide has been recommended but excess of this compound imparts a bitter taste to milk and, besides, commercial preparations of hydrogen peroxide are not always reliable. Ultra-violet rays have a destructive effect on bacteria, but application is more expensive than pasteurization.

Ozonization of milk is recommended by Wiener, who claims that all pathogenic bacteria are destroyed by the use of ozone and that no alteration in milk can be detected.

Pasteurization of milk may not be an ideal method of safeguarding milk supplies, but it is hard to escape the conclusion that under present conditions it is the best known remedy. Fortunately all the infections that experience has shown to be transmissible through milk are destroyed and the milk rendered wholesome by pasteurization. Pasteurization of milk is gaining rapidly in popularity and unless reliable methods are devised by which pathogenic bacteria can be effectually excluded pasteurized milk must be considered the most reliable milk produced.

THE SANITARY EXAMINATION OF MILK

The examination of milk consists of three phases, namely, the physical, the chemical and the biological.

Since physical, chemical and biological changes proceed rapidly in milk, it is of the utmost importance that representative samples be taken. If the milk is cold enough to solidify the fat, it should be warmed, otherwise the fat cannot be evenly distributed. This is especially true of cream, which forms a somewhat solid film when it stands for some time. Heating to 40 to 45°C. will melt the fat and make it possible to obtain a fair sample.

After warming the milk the sample should be mixed by pouring it several times from one vessel to another. Violent shaking is not advisable, because the air bubbles are retained rather tenaciously on account of the viscosity of the milk. If the sample is to be taken from a large volume of milk it should be stirred with a large spoon or one of the different sampling devices should be used. Milk samplers are tubes which reach to the bottom of the vessel and by a mechanical devise can be opened after immersing so that the column of milk removed represents exactly each layer.

Representative samples from sour and curdled milk are difficult to obtain. It is recommended that the casein be dissolved by addition of one-tenth to one-twentieth sodium or potassium hydrate solution or strong ammonia. Of course, this increases the volume and the results must be corrected accordingly.

Frozen milk must be thawed before sampling and unless the fat is liquefied 5 per cent. of ether may be added. The dissolved fat can then be distributed throughout the sample.

It is obvious that samples of milk should be examined as soon after taking as possible. If shipment is necessary or if it takes an hour or more to reach the laboratory samples should be packed in ice in order to inhibit changes in composition as far as possible.

In creameries and cheese factories, where producers are paid by the amount of fat delivered, it is customary to take composite samples. A definite volume is taken from each patron's milk and placed in a suitable container, such as a Mason jar. A preservative, such as mercuric chlorid, or potassium bichromate in the form of tablets, is added and the tests made once or twice monthly.

Physical Examination

The Specific Gravity.—Taking the specific gravity of milk was formerly thought to be sufficient for determining its quality. At the present time the specific gravity is considered of importance only in conjunction with other tests, since fraud which cannot be detected by this test is possible. The specific gravity of milk is due to the presence of solids. The dissolved solids increase it, while the fat decreases it. Milk rich in fat, therefore, has a lower specific gravity than milk poor in fat. A producer may partially skim his milk and thus increase its specific gravity, or he may remove some fat and then reduce the specific gravity to normal by the addition of water. The possibility of such fraud is, of course, limited, but by no means negligible.

The specific gravity of milk is usually determined by means of a lactometer. This instrument consists of a glass tube with graduations and thermometer, with a bulb at the lower end containing mercury. The milk is poured from one cylindrical vessel into another several times and then allowed to stand for about a minute. By this time air bubbles and foam have disappeared and the lactometer is immersed. Simultaneously the temperature is noted. The "Quevenne" lactometer which is graduated from 15 to 40 is the one most commonly used.

Another style of lactometer is known as the New York Board of Health lactometer. This differs from the Quevenne lactometer in that it has smaller degrees in its graduation which renders accurate reading easy. A third style is that devised by Shaw and Eckles. This one has a thinner stem and a larger bulb than the other forms and gives more accurate readings.

As there is some capillary adhesion of the milk to the stem of a lactometer care must be taken to allow for this. Either the surface of the milk must be taken as indicating the true reading, or, if the highest part of the adhering milk is read, 0.2 must be added to the reading.

The lactometer reading must be corrected for a variation due to temperature. Usually 60°F. is considered the normal. If the temperature is higher than this the specific gravity is lower; if the temperature is below 60°F. the specific gravity is higher. Tables for detailed correction are published in books dealing with the examination of milk, but fairly accurate temperature corrections can be made by deducting 0.001 for each degree below 60°F. and adding the same figure for each degree above.

For delicate work the Westphal balance or the pycnometer can be used. The Westphal balance consists of a plummet with a thermometer which is immersed in a glass cylinder containing the milk. The plummet is hung on the end of a balanced beam and by placing weights on the beam the specific gravity can be accurately determined.

The pycnometer is essentially a bottle with a glass stopper. The bottle is weighed accurately, filled with distilled water, the stopper inserted so as to squeeze out any excess of water, and the bottle filled with water weighed. The difference between the full and empty bottle gives the weight of the water. The water is then poured out and the bottle filled with milk, the latter weighed again and the specific gravity computed from the weight of the water and the weight of the milk.

The Sediment Test.—The sediment test has been used widely during recent years and has proved to be of great value. A definite quantity of milk, usually one pint, is filtered through a pledget of cotton upon which the insoluble dirt is gathered. Standards are made up upon similar pledgets of cotton and the sediment from a pint of milk compared with them. A fairly accurate quantitative estimate can thus be made.

This test accounts for the insoluble dirt only and is therefore not scientifically accurate. The dirt, chiefly cow manure, which enters milk contains variable amounts of soluble material. During the summer the soluble dirt is greater as a rule than during the winter. The test, however, shows approximately the amount of dirt in milk and serves as a valuable criterion of the methods used by the producer. Many large dairies test the milk of their patrons systematically and start a rivalry between producers which has helped much towards improving milk supplies.

The taste and odor of milk may be included in the physical examination, although little reliance can be placed on such tests unless pollution is heavy.

CHEMICAL EXAMINATION

The chemical examination of milk comprises chiefly the determination of fat, total solids and plasma solids. In addition to these tests there are several others which the analyst is sometimes called upon to make, chiefly a quantitative test for acidity and for milksugar. Further tests, made more rarely, are determinations of total nitrogen, casein and albumin.

Determination of Fat.—Fat is usually estimated by the centrifugal method devised by Babcock. This test is relatively easy to perform, even by untrained hands, and has consequently done much to elevate the dairy industry. This test makes it possible for dealers and creameries to determine accurately the amount of fat contained in the milk, and the custom is becoming quite general to pay producers for the fat delivered rather than for the weight of the milk. The advantages of this kind of remuneration are obvious; not only that no benefit can be derived from watering milk, since the actual amount of fat remains the same no matter how much water is added, but that the owner of a valuable herd producing rich milk is paid more in proportion than the owner of a poor herd.

The Babcock test is made by measuring in a specially constructed pipet 17.6 c.c. of the milk to be tested and this is discharged into a Babcock test bottle. The amount of milk discharged is not quite 17.6 c.c. as experience has shown that about 0.1 c.c. adheres to the pipet. Taking the average specific gravity of milk as 1.03 and multiplying this by 17.5, 18.025 or 18 gm. result. The Babcock test bottle is constructed with a long neck which is graduated in such fashion that 2 c.c. of the lumen are divided into 10 parts. By centrifugation the fat is carried into the range of this graduation. If the 17.5 c.c. of milk contained 2 c.c. of fat the proportion would be as 1.8 : 18 or 10 per cent., because 17.5 c.c. of milk weigh 18 gm. and 2 c.c. of fat, whose specific gravity is 0.9, weigh 1.8 gm. Therefore, each subdivision of the graduation represents 0.2 per cent. of fat. Recently Babcock test bottles have been constructed which have a graduation enclosing 1.6 c.c. and are graduated to 0.1 per cent. The reading of these bottles is of course more accurate than that of the earlier types.

After the milk has been placed in the test bottle, 17.5 c.c. of sulphuric acid (commercial specific gravity 1.82) are added. The sulphuric acid serves two purposes, namely, the complete solution of all colloids and the generation of sufficient heat to liquefy the fat. The fat is then easily collected in the neck of the bottle by repeated centrifugation.

The column of fat forms a meniscus at the top and at the bottom. Experience has shown that, in order to obtain accurate results, the precentage must be read from the top of the upper meniscus to the bottom of the lower meniscus. This is done because a small amount of fat adheres to the bottle and this loss can be compensated for by the given method of reading.

The Babcock test is also used for determining the fat content of cream and skim milk. For cream special bottles with wide necks and fine graduation are used because of the large amount of fat present. For skim milk or buttermilk the neck of the bottle has to be very narrow on account of the small amount of fat. The lumen of the neck is so small that it would choke were the milk and sulphuric acid poured through it. A second wider neck is, therefore, provided which reaches to the bottom of the bottle so that the fat can collect in the narrow neck.

For delicate chemical analysis the fat is determined by refraction or by ether extraction.

The Determination of Total Solids.—Total solids are determined by evaporating a definite amount of milk, usually about 5 gm., to dryness on a water-bath. After the water has been driven off the dish containing the residue is placed in a desiccator and the weight determined after complete desiccation. Some chemists add pure dry sand to the milk before evaporation.

Plasma solids or solids-not-fat are estimated by deducting the percentage of fat from the total solids.

There is a definite relation in milk between the specific gravity, fat and total solids, and when two of these factors are known the third can be calculated. The Babcock formula for calculating plasma solids is this:

$$\begin{array}{c}\text{per cent. plasma solids}\\ \text{(solids-not-fat)}\end{array} = \left(\frac{100S - FS}{100 - 1.0753FS} - 1\right) \times (100 - F)2.5$$

S is the specific gravity, F the percentage of fat.

Approximate estimates of plasma solids may be obtained by the following formula:

$$\text{Plasma solids} = 0.25L + 0.2F$$

and total solids are calculated by the formula:

$$\text{Total solids} = 0.25L + 1.2F.$$

L is the Quevenne lactometer reading.

Tables which save the operator the work of calculating are published in various treatises.

The Determination of Ash.—The ash is estimated by igniting the total solids over a slow flame. The residue should be white.

The Determination of Total Nitrogen, Casein and Albumin.—The total nitrogen is estimated by the Kjeldahl or Gunning method. Casein and albumin can be determined by several methods, of which the following is one of the simplest and most satisfactory. Twenty cubic centimeters of milk are diluted to 200 c.c. with distilled water and a few drops of a 10 per cent. solution of acetic acid added. Precipitation is complete when the casein settles rapidly and the supernatant fluid becomes clear. Excess of acid must be avoided as this redissolves the casein. The precipitate is collected on filter paper which has been dried and weighed, then washed with ether to remove the fat and with water to remove all soluble material. Finally the precipitate is dried and weighed. Or the nitrogen of the dried precipitate is estimated by the Kjeldahl or Gunning method. The amount of nitrogen is multiplied by 6.38 to express it as casein.

The filtrates are mixed and boiled to precipitate the albumin. The precipitated albumin is treated in the same manner as the casein.

A rapid method of casein determination, especially useful for cheese makers, has been devised by Hart. Two cubic centimeters of chloroform are placed in a specially constructed bottle and 20 c.c. of a 0.25 per cent. acetic acid solution added. Exactly 5 c.c. of milk are then measured into the bottle and the contents shaken. The bottle is placed in a centrifuge and whirled for seven and one-half to eight minutes at the rate of 2000 r.p.m. The bottle is set aside for 10 minutes and the casein allowed to form a clearly defined column in the graduated part of the bottle. The percentage can be read off directly.

The Determination of Milksugar.—Milksugar is estimated either by the reduction of copper salts or by the polariscope. The reduction of copper salts can be determined by titration with Fehling's solution. A definite amount of milk, say 20 c.c., is diluted 20 times with water, the casein precipitated with acetic acid and the albumin by boiling. The precipitated casein and albumin are washed with water to remove the milk-sugar completely. The clear fluids are mixed, measured and a portion filled into a burette. From this burette the solution is run into 20 c.c. of boiling Fehling's solution previously diluted with 80 c.c. water. Each 20 c.c. of Fehling's solution completely reduced represent 0.135 gm. of lactose.

The copper oxide precipitate from Fehling's solution with milksugar can be filtered off, dried and weighed. The amount of milksugar present is then calculated according to published tables.

The Determination of Acidity.—The acid found in milk is an index of its age, although there is not a clearly defined relation between the quantity of acid and age. That is to say, it is not possible to calculate the exact age of milk from its acid content, but an approximate estimation of value to consumers as well as to dealers and butter makers can be obtained.

Fresh milk reacts slightly acid to phenolphthalein, but this is due to acid phosphates and carbon dioxid and not to free acid. To litmus the reaction of fresh milk is amphoteric, that is to say, acid salts turn blue litmus red, while alkaline salts turn red litmus blue.

The acid in milk is measured by using normal alkali solution with phenolphthalein as indicator. Fresh milk should not show more than 0.2 per cent. acid, expressed as lactic acid.

Several rapid methods for estimating the acidity of milk have been devised of which the Farrington alkaline tablet test has become very popular. Each tablet contains enough alkali to neutralize 0.035 gm. lactic acid. A small amount of phenolphthalein is added to serve as indicator. The test is made by dissolving five tablets in 97 c.c. water; this is run from a burette into 17.5 c.c. milk, which are measured in a Babcock pipet. Each cubic centimeter of the tablet solution neutralizes 0.01 per cent. acid, and therefore, the percentage can be read directly from the burette.

Tests to Distinguish Human and Cow's Milk.—It is sometimes advisable to make a test to distinguish human milk from cow's milk. The immunological reactions previously mentioned furnish the most delicate test of this sort. There are also chemical tests, but these are not nearly as delicate. When human milk is mixed with ammonia and kept at room temperature it develops a reddish-violet color, whose intensity is proportionate to the quantity of ammonia used. Cow's milk does not develop this color with ammonia. Another method is carried out by mixing equal volumes of human milk with a 1 per cent. solution of silver nitrate. The mixture is heated to the boiling point at which a brownish-violet color develops. Cow's milk treated in the same manner develops a very faint color or none. A third test can be made with a 1 per cent. solution of neutral red in 0.8 per cent. sodium chlorid solution. A few drops of this solution in human milk produce a yellow color, while in cow's milk a red color is obtained.

The Biological Examination of Milk

The biological examination of milk consists of a microscopic and a bacteriological test. The microscopic test is made by staining a portion of the milk, usually with nethylene blue, and counting the cellular elements and log-chained streptococci. The value and interpretation of this test will be discussed in another connection.

The bacteriological examination consists chiefly of enumerating bacteria and in some laboratories special tests are made for certain types of bacteria which are considered by some authorities as indicative of manurial pollution. The search for pathogenic bacteria is a difficult problem for various reasons. For instance, some pathogenic bacteria do not thrive in milk, although they may retain their vitality. The tubercle bacillus does not multiply in milk, but its presence can be detected by inoculation of guinea pigs. Other pathogenic bacteria, as the typhoid and diphtheria bacilli, do multiply in milk, especially when it is not properly cooled. But their munber is usually small in comparison with saprophytic bacteria and they are, therefore, difficult to find. When bacteriological methods do not reveal the presence of pathogenic bacteria, their absence is not proved and the milk may be infectious. Fortunately there are means of detecting infectiousness in milk and by these means results are obtained in shorter time than would be possible by laboratory methods. This subject will be taken up later.

Enumeration of bacteria in milk can be made by two methods, although the plate method is most commonly practiced. The other method consists of direct microscopic counts and is stoutly advocated by some authorities.

The plate method consists of mixing a definite quantity of the diluted milk with a tube of liquefied agar, and then pouring the mixture on a sterile petri dish. Or, better, the diluted milk is poured on the petri dish and the melted agar mixed with the milk on the dish. After the medium has solidified, the dish is incubated and the colonies counted after they have developed. Each colony is taken to represent one organism originally contained in the milk. A modification of the plate method is the "Little Plate" method of Frost. The milk is mixed with agar medium and the mixture spread on a microscopic slide. After six to eighthours incubation the individual bacteria have grown into small colonies which are counted under the low power microscope.

The direct microscopic method of examination is championed chiefly by Breed and Brew. One one-hundredth of a cubic centimeter of milk is measured in a specially constructed pipet and smeared on a slide so as to cover 1 square cm. After drying and dissolving out the fat with xylol the smear is stained with methylene blue, and using the oil immersion lens the bacteria are counted in a number of microscopic fields. The average of fields counted is then calculated and the results expressed in number per c.c. For details of technique the reader is referred to a publication of the American Public Health Association entitled "Standard Methods for the Bacterial Examination of Milk."

Experience has shown that when clean milk is examined by direct counting the number of bacteria is higher than by the plate method. When the number of bacteria reaches about one million per c.c. the direct method of counting gives results about 5 per cent. higher than the plate method. By the direct method no distinction is made between dead and living bacteria which fact may account in part for the difference. In the plate method the colonies are counted and it is unlikely that all colonies actually originate from a single cell. It is a well-known fact that bacteria in milk form clusters, chains, and filaments, especially in fresh milk, and consequently many colonies originate from more than one cell. The count by the plate method, therefore, is always lower than the actual germ content.

The plate method gives account only of living bacteria and of bacteria able to multiply on the medium used. Furthermore, as the count is made after an incubation period of two to five days, slowly growing bacteria may not have formed visible colonies by the time results are gathered. It follows then, that soil bacteria and anaerobes, for example, are not counted by the plate method as they do not grow under the conditions of aerobic plating.

A count of bacteria by the plate method must then be expected to run considerably lower than the number actually present. All those familiar with bacteriological technique know that culture media made up according to different formulas, as a rule, yield different results. Not only the actual composition of the medium, but its chemical reaction is of importance. It is, furthermore, known that the period and temperature of incubation are factors that materially influence the results. Therefore it is necessary that in all laboratories where milk examinations are made the same technique be adhered to if results are to be comparable. Any deviation from a well-recognized technique should be noted with the results. The technique recommended by the American Public Health Association is the most approved, although by no means perfect, method, and is subject to change as investigations throw new light on our methods. This technique, therefore, should be adopted by all laboratories.

It is hardly necessary to call attention to the fact that bacteriological technique is very delicate, and any slip, though it appear ever so slight, may lead to discordant results. Reliable milk examinations, therefore, should always be made by experienced workers and should not be intrusted to any one who lacks proper training and who does not realize the responsibility involved.

After all, the burden of responsibility rests with those who have to interpret the results of bacterial examination. To guard against error counts should be made in duplicate or better in triplicate and when dependable figures are obtained the question may be asked, what do they mean? If the production has been careless, much bacteria-laden foreign matter may have entered the milk and still the number of bacteria in the milk may not be excessive if the temperature during transportation has been low. On the other hand, if the original product was clean and the temperature during transportation too high, the number of bacteria may be large. Some idea of which part of the handling has been at fault may be obtained by counting acid-forming colonies separately. If these are numerous, transportation facilities probably were at fault; if small, the producer has probably been careless. But the differential count is by no means entirely reliable, as it is quite possible that many lactic acid bacteria entered the milk during production. This may be especially true when utensils are not properly cleaned and sterilized.

Buttermilk contains several hundred million bacteria per c.c., but it would be folly to condemn it on this score. In this case it is known that by far the majority of bacteria present are of the lactic acid type, and therefore harmless, but it well illustrates the point that numbers alone are not necessarily an index of the wholesomeness of the milk.

When there is a large number of bacteria present in milk and yet transportation facilities have been shown to be above reproach, infectious material in the milk supply may be suspected. This follows from the reasoning that a producer who is careless or ignorant may also through carelessness or ignorance permit infectious material to reach his product. Such material may be derived from the milker or from the cow.

But there is another important consideration. Aside from the menace of pathogenic bacteria, milk should be a clean food product. Even though there is no menace to health, the consumer has naturally an antipathy to milk, or any other food, that contains filth. Attention has been called to the frequency of cow manure in milk and this is both disgusting and unnecessary.

A bacterial count of milk is the most important part of a complete milk examination, for it may indicate danger and, therefore, serves as a guide to the dairy inspector. The inspector can follow the milk during its journey and find the source of trouble. His suggestions will correct the fault and a subsequent bacterial test will inform him whether his suggestion has been properly carried out.

Other Bacterial Tests

In some laboratories a test for the number of B. coli in milk is made on the assumption that large numbers of this group indicate heavy manurial pollution. This test has been abandoned by many workers, however, because B. coli is practically present in all market milk and large numbers may be due to growth as well as to initial pollution. It has been shown that there is appreciable growth of B. coli in milk at as low a temperature as 10°C. and interpretation of results therefore is difficult. It should be remembered, furthermore, that most bacteria in milk originate from pollution with cow manure so that the B. coli test would seem superfluous.

Weinzirl and Veldee have recently recommended a test for anaerobes in milk, one which they consider a measure of manurial pollution. Anaerobes do not multiply in milk under ordinary conditions so that, the authors argue, the number of anaerobes represents the original pollution. It would be interesting to carry on a large number of tests by this method to learn its true value.

The collection of samples for bacteriological examination requires extreme care. The milk must be mixed, since, as has been pointed out before, bacteria either cling to the fat globules and go into the cream, or settle with insoluble matter. Growth of bacteria must be guarded against as much as possible when examination cannot be made immediately after the collection of samples. When an hour or more has to elapse samples should be packed in ice, and if this is done Conn maintains there is but a slight difference in count even after 20 hours.

MILK AS A CARRIER OF INFECTION

It has been known for some time that milk and milk products may spread infection. Epidemics have not infrequently been traced to milk supplies and considering the enormous amounts of milk and milk products consumed, it is astonishing that there is not an even greater amount of disease communicated by this means.

Pathogenic bacteria, as previously pointed out, are difficult to detect in milk. The relative number of disease germs in milk is usually small and this fact renders it difficult to isolate them. Isolation of a specific organism is a slow process and may consume several weeks time. Most pathogenic bacteria, if they are able to grow in milk at all, do so but slowly if the milk is kept at a low temperature. Furthermore, as germ carriers are frequently the means of infecting milk, and as it is known that they may discharge the infectious material intermittently, a sample of suspected milk may be examined repeatedly before the germ is found. And finally, by the time a milk supply is suspected the particular milk at fault has been consumed and no sample is obtainable for examination.

It is clear that no dependence can be placed upon an examination of milk for disease germs. Epidemiologists have other means of tracing the cause and although the evidence is chiefly circumstantial it is no less reliable.

An epidemiologist investigating an epidemic will prepare a spot map which shows the exact location of each case which has been reported. He will make a house to house canvass, learn the source of milk used; of water used; and many other points. From the spot map he can usually decide at a glance whether the water or milk supply is incriminated, since usually a community has but one, or at best a small number of water supplies, while milk supplies are more numerous. If the cases of disease are spread with more or less regularity throughout a community, the water supply is probably to blame, while, if a milk supply is at fault, the cases are concentrated in a particular locality. After suspicion has been fastened on a certain milk supply, the sources of the milk are investigated and if a carrier is suspected a bacteriological examination of all persons concerned with handling the milk will confirm the diagnosis.

Milk borne epidemics are, as a rule, explosive in their onset, like water borne epidemics, but are confined in most cases to consumers of a particular supply. The number of persons affected is conditioned by the amount of milk consumed; by the susceptibility of the individual; and by the manner in which the milk is taken.

The amount of milk consumed influences the number of affected persons because the infectious material is naturally ingested in greater quantity when much milk is taken than otherwise. The susceptibility of the individual plays a role as in all other modes of infection. Finally, milk or cream taken in hot coffee or tea is affected by the heat of these beverages and many bacteria are destroyed thereby. When milk or cream are taken by the glass or on cereals, puddings, etc. the menace remains unaltered.

Pathogenic bacteria in milk may come from dairy attendants or their clothes; from bottles or utensils; from flies and dust; from domestic animals; and from the cows. The attendants may communicate infection from their hands. This is especially true of typhoid fever. A milker, for example, who is a typhoid carrier, may allow germs from his hands to pass into the milk. Tuberculosis, diphtheria and other diseases of the respiratory tract are communicated chiefly by persons coughing and sneezing while milking or otherwise handling the milk.

Bottles become a possible source of infection when they are taken from homes in which there is a communicable disease. Cans become, a source of danger when they are handled by carriers without being properly sterilized. Polluted water supplies used for washing utensils may also contaminate the milk supply.

Flies and dust may infect the milk, although these are probably unimportant sources of contamination. Domestic animals are known to be able to carry infectious material from house to house and may do so if the milk bottle left at the backdoor early in the morning, is unprotected from nosing dogs and cats. The cow becomes a source of infection when she is tuberculous or suffers from some other disease to which man is susceptible.

Diseases which may be communicated through milk and are derived from human beings handling it may be: Typhoid fever; dysentery; paratyphoid fever; diphtheria; septic sore throat; human tuberculosis; Asiatic cholera; and scarlet fever. Diseases that may be communicated from sick cows are: Bovine tuberculosis; foot and mouth disease; mastitis or garget; contagious abortion; anthrax; rabies actinomycosis botryomycosis; cowpox; milk-sickness; and gastro-intestinal disorders.

Typhoid and Paratyphoid Fever and Dysentery.—Typhoid fever epidemics have been repeatedly traced to milk supplies. The cause is usually a carrier, although there are cases when the fecal discharges of a typhoid fever patient were carelessly left in places from which communication with the milk was possible. Dejecta from human beings should never be used for fertilizing, as some of the manured soil may wash into the well, or be carried on the soles of those working in the field. Well water is frequently used for cooling milk and if it is contaminated the milk may become infected. Typhoid fever is one of the most frequent of epidemics transmitted through milk, although paratyphoid fever and dysentery may be communicated the same way.

Diphtheria.—Diphtheria bacilli may reach the milk if the milker who is a carrier sneezes or coughs. Infection may likewise enter the milk from the hands of a carrier. There is, furthermore, a grave possibility of the germs being carried in bottles from houses where diphtheria exists.

Dean and Todd found peculiar pustules on the teats of a cow and found that they were caused by diphtheria bacilli. The cow is not susceptible to diphtheria as human beings are and these pustules were probably caused by a heavy infection from a milker. Such cases are extremely rare.

Septic Sore Throat.—Reports of sore throat epidemics caused by milk supplies have been published for some years, but it is only recently that the disease has in some instances been clearly traced to milk supplies. It has been believed that septic sore throat was caused by a streptococcus in milk, these organisms having been derived from udder inflammations of the cow. The work of Davis, Smith and Brown, Krumwiede, and Valentine, Mathers and others has done much to show that septic sore throat is not usually caused by mastitis streptococci, but by strains of streptococcus which are located in the human throat and communicated to milk supplies. It is natural, with any throat disease, that contact plays an important role in the transmission of this disease. This has been clearly shown by Winslow and Hubbard in a report on an investigation of an epidemic of septic sore throat in New York State.

Human Tuberculosis.—It is not probable that human tuberculosis has frequently been communicated through milk, but the possibility cannot be denied. A tuberculous milker may communicate the bacilli through coughing and sneezing or by contact with his hands.

Asiatic Cholera.—As Asiatic chlorea is an intestinal disease the chances of its being transmitted through milk are not excluded, although extremely remote. Asiatic cholera is an uncommon disease in this country and the spirilla causing it do not live long in milk because they are very sensitive to acid.

Scarlet Fever.—Although scarlet fever has been traced to milk supplies the mode of communication is still in doubt, largely because the causal organism is still unknown. Probably bottles and utensils are the chief vehicles of this disease, or perhaps the clothes of employees, according to Swithinbank and Newman. There is no reliable evidence that the disease can be derived from cows.

Milk Poisoning.—This term is sometimes used where infants cannot digest milk although there may be no poison in the milk. Real cases of milk poisoning are probably due to the action of members of the B. enteritidis and B. paratyphosus group of bacteria.

Bovine Tuberculosis.—Bovine tuberculosis is probably the most important disease communicated from the cow. This disease is widely disseminated among milk cows, and market milk, unless pasteurized or boiled, not infrequently contains living, virulent tubercle bacilli of the bovine type. According to numerous investigations from 5 to 17 per cent. of raw market milk contains the living organisms.

The question of infectiousness of bovine tubercle bacilli for human beings has been so frequently discussed in publications that it seems superfluous to recite the details again. Suffice it to state that the admirable studies of Park and Krumwiede have conclusively proven that bovine tuberculosis is not negligible in milk supplies. According to these authors during the age below five years 23.21 per cent. of all cases of tuberculosis were caused by the bovine type of bacillus; between 5 and 16 years 23.53 per cent.; and over 16 years 1.27 per cent. Preventive measures are, therefore, imperative.

The reduction of tuberculosis among cattle is a very difficult problem. It is true that a most accurate guide for the recognition of the disease is to be found in the tuberculin test, but if all animals responding to this test were slaughtered it would mean a terrific financial loss and perhaps a milk famine. Furthermore, not an insignificant number of producers fail to recognize the accuracy of the tuberculin test and oppose its application vigorously. It is for these reasons that other methods of eradication of the disease have been recommended of which the Bang system is the most efficient. In order to carry out this system all animals are tested with tuberculin and reactors removed from the herd. Animals with advanced tuberculosis are slaughtered while those not so seriously affected are used for breeding, their milk is pasteurized and then serves as food for calves. By this system a healthy herd is gradually built up.

The Ostertag system is based on veterinary examination. Only those animals are removed that suffer from advanced tuberculosis. Obviously this system is not as successful as the Bang system.

Bovine tuberculosis is disseminated through contact with infectious material in the stable, or any other place where tuberculous animals are kept; through introduction of diseased animals into a sound herd and by feeding calves an unpasteurized milk from tuberculous animals.

Bovine tuberculosis is frequently disseminated through creamery and cheese factory by-products. The skim milk from creameries and the whey from cheese factories should be pasteurized before it is returned to the producers and used for feed.

Under present conditions the most reliable remedy for safeguarding milk supplies from becoming a menace is pasteurization.

Paratuberculosis or Johne's Disease is occasionally met with in dairy herds. The disease resembles tuberculosis in its symptoms and causes a rapid decline in the health of the animal. It is caused by an acid-proof bacillus. It has been, and probably still is, frequently confounded with tuberculosis. A preparation made from Johne's bacillus is used for testing cattle for the presence of paratuberculosis, similar to the testing with tuberculin for tuberculosis. The disease is probably of no significance for human beings.

Foot-and-mouth Disease.—Foot-and mouth disease is caused by an ultra-microscopic organism which is contained in the milk of affected animals. Children are very susceptible to this disease and as the organism causing it is easily destroyed by heat suspected milk should be pasteurized.

Mastitis.—Mastitis or garget is a very common inflammation of the cow's udder. By far the majority of cases are due to streptoccic infection, but staphylococci and B. coli sometimes are the cause.

Mastitis appears in two forms, the acute and the chronic. The acute form is easily recognized physically and it affects the milk by making it stringy or watery. Red blood-cells appear in the milk and a yellow sediment forms on standing. The taste frequently becomes bitter. The chronic form is not readily diagnosed since the milk supply is not seriously affected and the milk shows no important change.

In one of these two forms mastitis is so common among milk cows that some authorities do not hesitate to state that there is probably no herd of milk cows free from it. The disease is accompanied by the appearance of many cellular elements in the milk and the streptococci frequently form long chains. In typical mastitis the streptococcus assumes the so-called "picket-fence" (Stackett) form, that is to say the long diameter of the individual cell is at right angles with the axis of the chain.

The presence of cellular elements in milk has given rise to some misapprehension. It was believed that when they were present in relatively large numbers pus was indicated. For this reason the unfortunate term "pus cells" has been frequently assigned to them.

As a matter of fact, there is no milk in the market free from cellular elements and there is some reason to assume that they play a role in milk secretion. But even milk with large numbers of these cells is not necessarily pathological. Prescott and Breed have counted 10,600,000 cells per c.c. in milk of normal quality and Breed later reported finding 54,300,000 per c.c. of strippings in a milk that seem edeminently fit for human consumption.

Apparently it would be a mistake to condemn milk on account of large numbers of cells. It should be mentioned that the methods for estimating the number of cells are in a measure responsible for some misapprehension. Early methods yielded irregular results, largely due to the fact that the milk was centrifugalized and the cells in the sediment counted. Prescott and Breed have shown that an accurate estimate can be obtained only by taking an average sample of the whole milk without centrifugation.

The significance of cells in milk, it must be confessed, is not understood, but it is certain that their presence does not always signify pus. However, when a stain shows large numbers of cells accompanied by long-chained streptococci, especially of the "picket-fence" type, there seems to be reason for assuming the presence of acute mastitis in the herd from which the milk was derived. Therefore, smears of milk should be examined for cells and streptococci and, although it may be premature to condemn milk on microscopic finding only, such conditions would indicate that a physical examination of the cows be made.

It is true that the presence of cells and streptococci has not definitely shown that the milk may be the cause of serious injury to consumers. Such milk has been fed to guinea pigs without causing the slightest detectable injury. Still it would be unwise to use mastitis milk for human consumption, as the subject is by no means closed and such milk must be more or less distasteful.

Anthrax.—Anthrax has been communicated through milk to man in only one case, as far as the author is aware, and this case, cited by Ernst, seems not to be entirely beyond question. Dissemination of anthrax through milk is of course conceivable, since anthrax is a common disease among cattle, especially in Europe. But if only one questionable case is reported and if it is considered that an enormous amount of milk is consumed, the menace is so small as to be insignificant. Besides, the presence of anthrax bacilli in milk would rapidly change its appearance in such manner as to make it distasteful and appear abnormal.

Rabies.—Rabies occurs among cattle and milk may contain the virus, but since it is not contracted through the alimentary tract its consideration is not merited.

Actinomycosis and Botryomycosis.—The spores of Actinomyces are widely distributed on grain, hay, straw and similar material and no doubt they may enter milk. However, there is no actual case of actinomycosis caused by milk on record. Both actinomycosis and botryomycosis occasionally attack the cow's udder.

Cowpox.—The danger from cowpox lies in the fact that it may be transferred from calves used for vaccine preparation. Milk infected with cowpox is especially dangerous to children and should not be used.

Milk-sickness.—Milk-sickness is a disease confined to certain and often very limited locations. Cattle, horses, sheep, goats, dogs, perhaps hogs, and human beings are susceptible. Milk-sickness, "trembles," or "slows," is caused by a spore-bearing bacillus, B. lactimorbi, according to Jordan and Harris. The spores are not destroyed by pasteurization. The infection may be communicated through milk.

It is questioned by some authorities whether B. lactimorbi is the causative organism, at least in all cases. Sacket, for example, has been able to produce the disease in animals by feeding them with a poison prepared from white snakeroot. We are dealing either with different diseases which have a similar group of symptoms, or the etiology has not yet been firmly established.

Contagious Abortion.—Contagious abortion is a frequent disease among cattle and the cause of enormous financial loss. Bang discovered the etiological organism. Recent investigators claim that the causative organism is widely distributed and frequently found in large numbers in some milk. Miss Evans suggests that it is closely related to the bacillus of Malta fever.

It does not seem certain that B. abortus is pathogenic for man, but as long as uncertainty exists milk containing this organism should be pasteurized.

Gastro-intestinal Disturbances.—When cows suffer from gastrointestinal troubles the milk may assume a bitter taste, and become thin and bluish. Such milk may cause similar disturbances in man and should not be used.

Malta Fever.—Malta fever is caused by a small bacterium, Bacillus melitensis, and is communicated through goat's milk. It was first studied on the island of Malta where a large percentage of goats were affected, but it occurs also in other places. The disease is known to exist on the islands of the Mediterranean, in India Africa, the West Indies, the Philippine Islands and in America. The disease is often of long duration, but the mortality is low.

A close resemblance has recently been established between the organism causing Malta fever and the bacillus of contagious abortion in cows. The two organisms cannot be considered identical unless further studies bring conclusive evidence.

It may not be superfluous to emphasize the fact at this point that the only reliable way of rendering milk safe from the infections mentioned is pasteurization. It is true that pasteurization does not destroy the spores of anthrax and milk-sickness, but both diseases are so rare that special precautions can be applied when there is reason to assume the presence of these spores. Public health demands pasteurization to protect it against many ailments that milk may communicate.

HUMAN MILK

Human milk differs from cow's milk in many essential properties. Its color is more yellowish, and its taste, owing to a large percentage of milksugar, is sweeter than that of cow's milk. The fat globules are less in number but larger in size than those of cow's milk. Creaming is very rapid and in a few hours the fat has separated so completely that the skim milk is transparent. The cream of human milk is more yellow than that of cow's milk. The amount of fat in human milk varies in similar manner as that of cow's milk. The first milk drawn from the breast is poorer in fat than the last.

The casein of human milk is in a very fine colloidal state and coagulates with difficulty. Acetic acid produces a fine, delicate precipitate and rennet apparently produces no coagulation unless the reaction is made decidedly acid. Human casein is split by rennet into paracasein and a soluble protein. When human casein is dissolved in alkali it forms a yellow solution. According to a recent investigation of Bosworth and Giblin, human casein resembles the casein of cow's and goat's milk, is tribasic and has a similar molecular weight.

Human milk sours on standing much as does cow's milk. The reaction of human milk is but slightly acid to phenolphthalein and alkaline to litmus.

Hammarsten states that gastric juice coagulates human milk and then rapidly dissolves the coagulum. The same author gives the specific gravity of the fat of human milk at 0.966 and the melting point at 34°C.

Human milk contains less phosphoric acid and more lecithin than cow's milk. The percentage of ash in human milk is 0.19 to 0.34, against 0.7 to 0.9 in cow's milk.

GOAT'S MILK

Goat's milk is used in many countries and is considered by some authorities to have some advantages over cow's milk as food for infants. A goat produces more milk in proportion to her size than a cow and is sometimes called the poor man's cow.

The fat globules in goat's milk are small and consequently rise slowly. In fact the cream is difficult to separate even by a centrifugal separator. Goat's milk has a "goaty" taste, probably for the same reason that cow's milk has a "cowy" taste, owing to the presence of manure. The goaty taste is also characteristic of cheese made from goat's milk.

With rennet goat's milk forms a compact clot and may therefore be less digestible than cow's milk.

Bosworth and Van Slyke have studied goat's milk and state some interesting points in which goat's milk differs from cow's and human milk. Goat's milk contains tri- and di-magnesium phosphate; tri-calcium phosphate; and mono-potassium phosphate. Human milk contains no insoluble phosphates, while cow's and goat's milk do, but the latter two milks contain more phosphates than human milk. Goat's milk contains more chlorids than human and cow's milk.

The authors give the following comparison of the composition of cow's, goat's and human milk:

A Comparison of the Composition of Cow's Milk, Goat's Milk, and Human Milk

Compounds	Kind of milk		
	Cow	Goat	Human
Fat	3.90	3.80	3.30
Milksugar	4.90	4.50	6.50
Protein combined with calcium	3.20	3.10	1.50
Salts	0.910	0.939	0.313
Di-calcium phosphate	0.175	0.092	0.000
Tri-calcium phosphate	0.000	0.062	0.000
Mono-magnesium phosphate	0.103	0.000	0.027
Di-magnesium phosphate	0.000	0.068	0.000
Tri-magnesium phosphate	0.000	0.024	0.000
Mono-potassium phosphate	0.000	0.073	0.069
Di-potassium phosphate	0.230	0.000	0.000
Potassium citrate	0.052	0.250	0.103
Sodium citrate	0.222	0.000	0.055
Potassium chlorid	0.000	0.160	0.000
Sodium chlorid	0.000	0.095	0.000
Calcium chlorid	0.119	0.115	0.059

An advantage claimed by advocates of the use of goat's milk is that goats are not susceptible to tuberculosis. It is true that tuberculosis has been found rarely in goats, but it has been found and possibly if goats were herded together in insanitary stables, as cows have been for untold generations, they might be found to be more susceptible to tuberculosis than is suspected.

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CHAPTER XXII

BREAST FEEDING AND NUTRITION

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ANATOMY OF THE MAMMARY GLANDS

The breasts are modified cutaneous glands which are functionally closely associated with the reproductive organs. They have been adapted to the secretion of milk for the nourishment of the infant, and undergo marked structural changes at the time of puberty, during pregnancy, and during lactation. They are situated upon the ventral surface of the thorax, one on each side of the midline, and while subject to very wide variation, extend on the average from the second or third rib to the sixth or seventh costal cartilage and from the lateral border of the sternum to the axilla.

Their shape is conical or hemispherical, and their consistency firm and elastic. The left is usually somewhat larger than the right. The diameter varies from 10 to 13 cm., the long axis being parallel to the border of the pectoralis major muscle.

During childhood the size of the mammary gland remains relatively the same as at infancy, but at the time of puberty it undergoes a rather sudden and marked enlargement. This enlargement involves all parts of the gland but is especially participated in by the stroma and fatty tissues. At the subsequent menstrual periods there is usually a little additional enlargement and transient soreness. The vascularity is thought to be considerably increased at this time. Ordinarily the mature gland is from 12 to 15 times larger than before puberty. When pregnancy occurs the glands again undergo a period of growth, this time especially referable to the parenchyma. This growth continues after the birth of the child and the breast may be from two to three times as large when fully functioning as before pregnancy.

Structure.—Like other glands, the breast is composed of the essential epithelial structure or parenchyma, and the supporting and connecting tissues or stroma. Besides this it is covered with the ordinary layer of skin.

Parenchyma.—The parenchyma of each breast is a flattened, circular mass of glandular tissue of a whitish or reddish-white color which is thickest behind the nipple and thins out peripherally. The anterior surface is convex and irregular due to numerous pyramidal processes that extend towards the skin. The base is flat or slightly concave and much smoother. The circumference is well defined and thick but presents numerous irregularities which extend beyond the limits apparent from the surface.

The parenchyma or corpus mammæ is subdivided into from 15 to 20 separate lobes which are variable in size and pyramidal in shape, with their apices pointing towards the nipple, their bases towards the periphery of the gland. Each of these lobes has one excreting duct which opens by means of a contracted orifice in a depression upon the tip of the nipple. The ducts converge near the base of the nipple and run parallel to each other and in the direction of the nipple to the exterior. Each duct is large enough to be easily visible to the naked eye and measures about 1.5 to 2.5 mm. in diameter. Near their ends, under the areola, each duct has an ampulla-like enlargement, the lactiferous sinus, which may serve as a small temporary reservoir for the secretion. The ducts do not anastomose throughout their course but may have a common opening on the nipple. There are no valves, but when empty the lining falls into longitudinal plicæ.

Microscopically, the ducts present an external coat of white fibrous connective tissue containing both circular and longitudinal elastic fibres. The lining epithelium is cubical or columnar except near the orifice where it becomes stratified squamous and is contiguous with that of the skin.

Each lobe is divided into numerous lobules centering about divisions of the excretory duct. These consist of numerous alveoli, tubulosaccular, spherical, or pyriform, which are situated about the terminal branches of the duct. The alveoli are typically made up of a single layer of epithelial cells enclosed by a basement membrane. The alveolar cells are the true secretory cells of the gland. When the gland is active these cells are granular, polyhedral, or cuboidal in type, and so closely packed together as to leave almost no lumen in the alveoli. During lactation, various stages of secretory activity are found in the different cells. The central ends contain minute fat globules and are more or less flattened, depending upon the degree of distention of the alveolus.

The lobes, lobules, and alveoli, are completely covered by a white fibrous connective tissue sheath which is not a distinct capsule. Outside of this all of the structures are surrounded and embedded in the subcutaneous adipose tissue. On the ventral surface this layer is especially well developed and fills in between the irregularities of the parenchyma and gives the smooth appearance to the gland. In women who have never born children, but little fat is found between the lobules, but much more is present in multiparous women. During lactation this fat is absorbed to a certain extent and causes the lobules to stand out more distinctly. Beneath the areola and nipple, however, there is no fat and the connective tissue is loosely arranged to allow for free movement of the nipple and easy distention of the ducts and sinuses during nursing. Posteriorly, the gland is attached very loosely to the fascia of the pectoralis major muscle.

The size of the mammary gland gives no index as to its secretory ability, for it may be made up of a large amount of stroma with but little parenchyma, or contain but little stroma and consist largely of parenchyma. Accordingly, a large breast may produce but little milk while one that from the exterior appears to be small, will secrete large amounts.

The skin which covers the breast is unusually white and is covered with lanugo hairs that are associated with sebaceous glands. Many ordinary sweat glands are also present. The subcutaneous veins are often readily seen owing to the thinness of the skin. The skin is closely adherent to the subcutaneous tissue but permits of much stretching during the enlargement which occurs in lactation because of its flexibility and elasticity. Nevertheless, linea albicantes are frequently produced.

The **areola** of the breast is covered by a thin, delicate, and pigmented skin. In young women who have not borne children, it is reddish in color but varies somewhat with the complexion. During pregnancy a marked increase in the amount of pigment takes place, so that in brunettes the areola becomes distinctly black. Sometimes during pregnancy there is a second ring of pigmented skin known as the secondary areola. After lactation is over; the pigment disappears somewhat but never to the degree before pregnancy. Numerous irregular elevations are seen upon the surface of the areola due to the underlying large sebaceous glands and to the glands of Montgomery. Visible sebaceous glands are also present outside of the areola. All of these structures enlarge greatly during pregnancy and secrete substances which are discharged upon the surface of the skin. Under the epithelium there is no fat but a well developed layer of smooth muscle is present. The fibers intercross in various directions but are mainly circular and radial. They are most numerous near the nipple where they often form a layer 2 mm. thick, and become continuous with the smooth muscle of the nipple.

The **nipple** shows a wide individual variation in position, but in most cases is situated slightly below and laterally to the center, of the breast and on a level with the fourth rib or fourth intercostal space, and about 12 cm. from the mid-sternal line. It is conical or cylindrical in shape, about 10 to 12 mm. in length, and has a rounded, fissured top with the openings of the lactiferous ducts upon it. The skin is like that of the areola except that there is no pigment on the top of the nipple. In the corium of the nipple are many papillæ, rich in nerves and vessels.

There is practically no fat present. Great numbers of sebaceous glands are found here, whose secretions tend to keep the skin soft and protect it against the saliva of the infant. The muscular layers of the nipple are continuous with those of the areola and consist in a marked external circular layer and a less distinct longitudinal layer, running parallel to the lactiferous ducts. There are also numerous irregular muscle fibers and elastic fibers together with loose connective tissue but no fat. During pregnancy the nipples increase in size and become more easily erectile.

Typically the nipple is conical or cylindrical in shape, but it may be flattened, somewhat pedunculated, discoidal, or hemispherical. Occasionally, the nipple will be completely inverted and may or may not appear at the surface upon stimulation. It is said that the musculature of the nipple by producing intermittent rhythmic contractions, has a tendency to empty the milk ducts, or to act as a sphincter when firmly contracted. These contractions make the nipple firm and erect when stimulated.

Blood Supply.—The mammary gland receives most of its blood supply from the anterior perforating branches of the internal mammary artery. Those passing through the second and third intercostal spaces are especially large. The lateral thoracic artery also sends some branches to the lateral and inferior portions of the breast. Minor branches are also received from the thoraco-acromial arteries, and from the anterior branches of the lateral cutaneous rami of the aortic intercostal arteries on the deep surface.

There is a free anastomosis between these vessels which form a loose network in the stroma on the surface of the breast. From this network branches extend around the lobes and lobules, and terminate as a fine network of capilliares about the alveoli. The venous capillaries arise from these and form two groups which follow the course of the arterioles, deeply and superficially. The superficial group does not follow the arterioles throughout but forms a loose network beneath the skin, which becomes very prominent during lactation. They become continuous with the superficial veins of the neck, abdomen, and with the thoraco-epigastric vein. The deep group unites and enters through large branches into the subclavian, intercostal, internal mammary, and axillary veins. Sometimes the superficial group may connect with the internal jugular and femoral veins.

The Lymphatics.—The breasts have extremely numerous and freely anastomosing lymphatics, the origin and distribution of which are not clearly understood. There is, however, an extensive network beneath the skin and areola which empties into the subareolar plexus. The lymphatics arising from the alveoli enter for the most part into the same plexus. From the subareolar plexus there are usually two large lymphatics that arise and pass laterally and empty into the axillary lymphatic glands. Other lymphatics are found following the course of the blood-vessels.

The Nerves.—The parenchyma of the gland receives its ennervation from the lateral mammary rami of the anterior branches of the lateral cutaneous branches of the fourth, fifth, and sixth intercostal nerves, and from the medial mammary rami of the anterior cutaneous branches of the second, third, and fourth intercostal nerves.

Some sympathetic fibers enter the gland with the blood-vessels. Their function is the control of the smooth muscle fibers of the breast but have little influence upon its secretory activity.

PHYSIOLOGY OF LACTATION

Preparation for Lactation.—As mentioned in the preceding section, the mammary gland is a rudimentary structure until the time of sexual maturity, when, under the influence of the secretions of the ovary, it undergoes a marked growth. Little change occurs in the parenchyma of the gland, however, until after pregnancy, when a very profound change takes place. The rudimentary ducts and epithelial cords rapidly proliferate to form the lobes and lobules of the mature gland; and the blood-vessels of the breast enlarge and multiply, greatly increasing the blood supply. Pigmentation of the nipple and areola takes place early in pregnancy together with enlargement of the sebaceous glands and follicles of Montgomery in the same area. Enlargement of the whole organ is usually marked. Shooting pains and other sensory phenomena are frequently noted. Before the sixth month of pregnancy the histologic structure is practically that of the completely developed gland. A watery secretion, sometimes profuse, is frequently present as early as the third or fourth month.

In the multiparous glands there is a similar but less marked change. At the completion of lactation there is some resolution of the glandular structure, the pigmentation of the areola becomes less evident, and a reduction in size takes place. With the advent of another pregnancy a reversal of these processes occurs and the secretory cells again prolifferate to form new alveoli, and the gland as a whole increases somewhat in size. The essential factors involved in the preparation for lactation are proliferation of the parenchyma and increased blood supply.

Relation between Milk Secretion and the Internal Secretions.—The appearance of milk in the mammary glands about three days after delivery, whether the pregnancy has been completed or prematurely interrupted, has lead to studies which indicated the influence of some internal secretion or hormone in stimulating lactation. However, very little is actually known concerning the nature of this relationship, and we can hardly expect to know much about the effect of the glands of internal secretion upon lactation until we know more about those glands themselves. Nevertheless some observations are very significant. Transplanted mammary glands have been observed by Knoepfelmacher[2] to secrete milk subsequent to delivery. It is known that the mammary glands never develop in animals that have their ovaries removed before maturity and that atrophy of the mature gland takes place when the ovaries are removed from adults. The growth of the breasts ceases and resolution commences with the intra-uterine death of the fetus. Starling[3] has shown that removal of all the internal genitalia, including the uterus and fetus, from a pregnant dog causes lactation to commence. He concludes that it is the removal of some element from the maternal organism that inaugurates lactation. He has further been able to cause growth and secretion in the mammary glands of rabbits by injections of fetal extracts. From this he concludes that during pregnancy the fetus secretes some substance which causes the mammary gland to hypertrophy but which inhibits secretion and causes a storing up of energy. With the removal of the fetus and of the inhibition, this process is reversed and secretion takes place. However, this theory does not explain certain other phenomena, particularly the secretion which usually takes place from the breasts of newborn children at the same time as in the mother.

Furthermore, similar enlargements and secretion from the mammary glands has been seen to follow the injection of various other tissue and glandular extracts. The transplantation of the ovary of a pregnant animal into a virgin has caused the growth and subsequent lactation of the mammary glands. Injections of corpus luteum obtained from gravid cattle produce similar effects. Preparations of the placenta are reported as having like action. Hammond[4] suggested that the corpus luteum causes the development and secretion of the mammary glands and that the placenta produces a substance inhibitory to mammary secretion. Thus during pregnancy growth and development take place but secretion is inhibited, but with the expulsion of the placenta the inhibition is removed.

All of these theories are still very inadequate, and the subject is becoming increasingly complex. There are few things which would influence infant feeding more favorably than the solution of the physiologic principles controlling lactation, but at the present time nothing is known which can be of any practical value in the regulation breast feeding.

Effect of Nervous System on Lactation.—It has been fairly definitely established that the nervous system has but little influence on the mammary glands. Mammary glands have been transplanted from one location to another on an animal and observed to function later. All nerves leading to the milk glands have been divided without in any way influencing the secretion. There is a record of a pregnant woman who had her lumbar cord completely destroyed but who later gave birth to a child and lactated in a perfectly normal manner. One of the most interesting observations is that of Basch.[5] He reports the pygopagous twins (Blazek sisters) whose nervous systems were separated but whose circulation was common. One became pregnant and gave birth to a child while both lactated in an apparently normal manner. These observations lead us to believe that the nervous system has nothing to do with inaugurating the function of lactation. The work of Schaffer and McKenzie[6] and of Starling[7] in which they tried stimulation of the nerves leading to the mammary gland by all possible means, further indicates that these nerves are not related in any important way to milk secretion, after the function has been established. They were unable to produce any effect by such stimulation.

It is frequently stated, and apparently seems true, that some women's milk is influenced by nervous factors. The above physiologic facts do not tend to bear this out, however and it is probable that the effects are produced indirectly. Nervous and psychic disturbances in the mother may readily bring about a reduction of milk supply if they cause reduced food or fluid intake or if they interfere with her rest. Even very young infants quickly reflect their parents, state of mind and under unusual nervous influences, may become fatigued and nurse very poorly. Such factors no doubt play an important rôle in nursing but there is no physiologic evidence that the effects are produced directly through the nervous system.

Effect of Sucking on Milk Secretion.—While various hormones and internal secretions are undoubtedly active in preparing the breasts for lactation and in starting that function, there is no doubt but that by far the most important factor in the maintenance of the secretion is the act of sucking. We shall take up in detail in a later section the methods of restitution and maintenance of lactation but at this point would like to refer to some of the physiologic principles upon which those methods are based.

The supply of milk is determined by the demand which is made upon the breast. A breast which produces a large amount of milk will quickly reduce its output if but partially emptied when nursed. On the other hand a breast which yields but a small amount will almost as quickly increase its output if regularly and completely evacuated. It has been said that the normal function of the mammary gland is to produce milk at all times, failure to do so resulting from lack of demand for it. Pfaundler[8] and more recently, Fink, have reported calves who were sucked by other calves with which they were penned. In a short time they began to give milk and before they were a year old were giving over a quart a day of normal milk. Nursemaids who have made a practice of putting crying babies to their breasts to still them have been known to begin to secrete considerable quantities of milk although they themselves were virgins. Cases of the same thing occurring in the male are known. It is a frequent experience for milk to return to a mother who has weaned her babe months before, after simply placing him back at the breast. Breasts that are never nursed following delivery do not secrete milk for more than a few days. To our minds, the physiologic factor which is most important in the production of milk is the act of sucking. This is very fortunate and especially important, because it is the factor over which we can exercise the greatest control. Through creating the proper demand on the breast, we can in all cases produce some breast milk for every child, at least for a time.

Galactagogues.—A great many substances have been employed as galactagogues from early times. Scientific investigations, however, have shown that few, if any of these are of any benefit. Practically no drugs have any effect upon mammary secretion. Atropin and pilocarpin have no constant or marked influence.

Recent investigations with tissue extracts and internal secretions have shown some relationship between other organs and the breasts. Shaffer and McKenzie,[9] Hammond,[10] Ott and Scott,[11] and others have investigated the influence of pituitary extract (posterior lobe) and corpus luteum on lactating animals. All are agreed that there is a temporary increase in milk production following their administration. The effect never lasts for more than 24 hours however, and often for as short a time as 10 minutes. A diminution of the secretion often occurs afterwards so that the average is not changed. Gavin[12] has been unable to produce any changes in the milk production of cows by the use of pituitary extracts. Extracts of placenta, fetus, adrenals, thyroid, ovaries, etc., have been employed by many with widely varying results. Some investigators assert such extracts increase the secretion, while others claim the same extracts decrease milk secretion. Most workers have concluded that there is as yet no definitely known action for any of these substances.

Wolf[13] has strongly advocated the subcutaneous injection of milk as a galactagogue but Chatin and Rendu[14] repeated his work and were unable to find any effect. This work has more recently been repeated with negative results. The blood of lactating animals does not contain any galactagogue, at least in amounts sufficient to produce any effect on transfused animals. We, therefore, have no drug or biologic preparation which is of any value in the production or increase of mammary secretion, and we must rely on the physiologic methods described later in this work.

Composition of Human Milk.—Breast milk varies widely in appearance. In general it resembles cow's milk but is rather paler and likely to contain flocculent particles. Specimens with high fat content, however, may be very yellow. It is distinctly sweeter than cow's milk and is odorless. Microscopically, the tiny fat droplets which are held in suspension are seen. Occasional leukocytes and epithelial cells are also present.

Freshly drawn human milk is amphoteric in reaction due to the presence of both mono- and diphosphates. The specific gravity varies between 1.020 and 1.035, the average being about 1.030.

As the milk leaves the breast it is practically sterile, although Staphylococci can be obtained from most samples. This is due to contamination from the nipples and larger ducts and bears no relationship to the health of the infant.

Chemically, milk is composed of all the elements of the body, namely, protein, fat, carbohydrates, salts, water, extractives, and vitamins. Citric acid is also said to be present in small amounts. A detailed discussion of the characteristics of these substances will be found in other portions of this volume and in special treatises. The protein occurs as casein, lactalbumin, and lacto-globulin. Casein forms by far the largest part of the protein present and as far as we know is the only protein of importance in infant feeding.

About one-half of the non-volatile fatty acids are oleic acid. Myristic, palmitic, and stearic acids make up the balance. Volatile fatty acids are present but in much less quantity than is found in cow's milk. Butyric, capronic, caprinic, and caprylic acids are known to be present.

The carbohydrate occurs as lactose only. This is the same as is found in the milk of lower animals.

The salts in human milk are shown in the following table:

Composition of Ash of Human Milk (From Engel)

	100 gm. milk contains, milligrams			100 gm. ash contains, milligrams		
	First milk	End milk	Average	First milk	End milk	Average
K_2O	100.8	63.4	88.4	32.5	31.9	32.4
$B_{22}O$	44.8	17.6	35.7	14.5	8.9	13.1
Na_2O						
CaO	37.6	38.1	37.8	12.1	19.2	13.9
Mg.O	5.4	5.2	5.3	1.7	2.6	1.9
Fe_2O_3	0.22	0.12	0.2	0.07	0.00	0.07
P_2O_5	32.10	28.8	31.0	10.40	14.50	11.40
SO_3	9.6	7.2	9.0	3.1	3.6	3.3
Cl	71.7	34.2	59.1	23.1	17.3	21.7

There is considerable variation in the different elements of the milk but the variation in the fat content is by far the greatest. The protein and carbohydrate do not vary sufficiently to be of recognized importance in feeding. It is interesting to note that when one element is present in excessive amounts, the others are usually somewhat decreased. For all practical purposes the average composition of human milk is as follows: Fat 4 per cent., lactose, 7 per cent., protein, 1.5 per cent., salts 0.21 per cent. Very wide fluctuations from this, however, are not to be regarded as abnormal.

Effect of Diet on Composition of Milk.—Although considerable investigation has been made, we are as yet unable to draw any definite conclusions concerning the influence of diet upon the quantity or quality of breast milk. Czerny and Keller's conclusion that the supply of breast milk cannot be permanently influenced by the diet still stands. Variations in the fat content occur temporarily after fat feeding but the normal percentage is quickly restored. Starvation reduces the quantity of milk but Eckles has shown that in cattle the fat percentage is then greatly increased. It is important to note that the intake of large quantities of fluid has little influence on milk production for this method is still extensively used in attempting to increase milk supply.

Effect of Pregnancy, Menstruation, Etc.—Many pediatrists recommend weaning with the advent of pregnancy because of the supposed inability of the mother to nourish herself and two children. There is absolutely no foundation for such a conclusion because we have frequently seen wet nurses who nurse two and even three children perfectly well. No changes in the composition of the milk after pregnancy have ever been demonstrated and our clinical observations certainly do not indicate such change.

Menstruation also has no important influence on the composition of breast milk. Examinations before, during, and after menstruation show practically the same figures for all elements. Clinically, there is no question, however, concerning the influence of menstruation on the child, but as said before this is temporary, insignificant, and never a cause for weaning.

Excitement, sorrow, nervousness, etc. have been repeatedly quoted as causes for the reduction or disappearance of the milk. No laboratory experiments are on record to substantiate any such phenomena although they do seem to occur clinically. We are of the impression that the clinical findings are indirect and that the real cause of such reductions is to be found in disturbed habits and methods of nursing.

Excretion of Drugs in Milk.—Talbot and Morse give the following list of drugs which have been proved to exist in the breast milk in small quantities after ingestion by the mother.

Potassium iodid, sodium salicylate, antipyrin, mercury, aspirin, calomel, arsenic, bromids, urotropin, and certain fat soluble substances.

Alcohol occurs only after drinking excessive amounts. Opium and atropin have been demonstrated in the milk of animals but not of human beings.

Salvarsan is excreted in considerable amounts after the injection into the mother. This constitutes an important method of procedure in the treatment of congenital lues.

FEEDING IN THE FIRST FEW DAYS OF LIFE

When to Put Child First to Breast.—The process of labor is a strenuous ordeal for the mother and the child. Both of them have been subjected to severe physical exertion for many hours before the function is completed, and naturally need sleep and rest. Most newborns, after being cleansed, dressed warmly, and placed in their cribs, will sleep quietly for nearly 24 hours. If they awaken and cry, usually a change of the diaper is all that is required, after which they resume their sleep. Many authors recommend that at such occasions the infant be given water, water or tea sweetened with saccharin, or even catnip tea. The apparent purpose of these administrations is to calm the child, but such measures probably are not necessary and the requirements of the infant organism for water at this time will be considered in another place.

Talbot and Morse recommend first placing the child at the breast 6 to 12 hours after birth and every six hours during the next day, the third day every four hours and when the milk comes in at shorter intervals. Their purpose in putting the infant to the breast so soon is not to give it food, but to stimulate the secretion of the milk. All pediatricians are not in accord on this subject. Czerny for instance, advises that the child be put to breast, not at all on the first day, once on the second, twice on the third, increasing thus once each day until the child receives five feedings on the sixth day and thereafter. Feer recommends leaving the child off the breast until it evidences hunger, and states that seldom does this manifest itself before 24 hours or usually longer, even up to 48 hours. The latter views are probably more in accord with Taylor's[15] findings. It has been a custom of many to use an intermediary method, putting the child to breast once towards the end of the first 24 hours and then to go directly on with a permanent feeding schedule.

Frequency of Feeding in First Few Days.—Some pediatrists advise nursing the infant at two- to three-hour intervals during the first few weeks, others, with apparent success employ the four-hour schedule from the establishment of lactation. Our experience leads us to believe that except in rare instances the child thrives, gains weight, and sleeps well, under this method. On the other hand we have, in so doing, taken an important prophylactic measure against overfeeding, a not infrequent occurrence in the newborn when the short interval is used, and have also relieved the mother somewhat from the strain of nursing.

Difficulty in Getting the Newborn to Nurse.—There is seldom any difficulty in getting a full-term infant to nurse. Doctor Litzenberg, who has had a large obstetrical practice in Minneapolis for years, tells us that he has seen only one infant that would not nurse well. Many when placed at the breast for the first time grasp the nipple at once and nurse with strength and vigor; others are slow to grasp it and then give only a few tugs and let go again, repeating this each time the nipple is placed in their mouths. Some show a tendency to sleep and must be repeatedly shaken or aroused to be kept awake. It is not well, however, to try to force a child. If after a fair trial for 10 to 15 minutes, there is no attempt made to nurse, he should be placed back in his crib and not disturbed again until the next regular feeding time. An error is sometimes made by placing the child at breast when it awakens and cries an hour or two later. This is no excuse for breaking up the child's schedule and may easily result in starting irregular habits. The feeding of the first few days of life is more important than that of any other time in the establishment of regular feeding and sleeping habits A child spoiled at this time, may take weeks to become accustomed to the desired schedule later. If the infant is normal, a fact which must always be carefully determined, he should continue to be put to breast at the regular intervals and every attempt made to assist him. Very soon he will become hungry enough to be glad to nurse. It is sometimes of help to express a few drops of milk from the nipple into his mouth so that the taste of the milk will attract him. The practice of putting sugar on the nipples does not seem a good one.

It is occasionally reported that infants are "afraid of the breast" and will nurse well only when blindfolded or in a dark room. Such cases are surely very rare if they occur at all, but as mentioned above one must be sure that there is no physical reason why the child is slow to nurse. Usually such obstacles as deformities of the lips and mouth, nasal obstruction, retracted or small nipples; too large, or cracked nipples, are obvious; but prematurity, or cerebral hemorrhage may be easily overlooked.

Should Water Be Given?—The widest variation of opinion exists relative to the administration of food other than breast milk, and of water during the first few days of life. Authors rather generally recommend water, either in spoonful amounts, by the ounce, or as desired, until lactation is established. This is supposedly given to prevent dehydration, uric acid infarction, and hunger. Recent observation, however, has led many pediatrists to believe that while this water can do no harm, it is often unnecessary. At the University Hospital during the past two years, we have observed a series of 700 successive newborns, one-half of whom were given water ad libitum, after each nursing, starting 16 hours after birth, and the other half of whom received nothing but what they obtained from the breast. These children were taken alternately so that the two groups are entirely comparable. A careful analysis of these cases has been made and it could not be determined that the administration of the water produced any beneficial or detrimental effect. The initial loss of weight averaged the same. No differences in temperature were evident. "Inanition" or "dehydration temperature" occurred with just as much frequency and severity in the one as in the other group. Urinary secretion was increased in the babies given water, but the other group showed no abnormalities in this respect. The time of appearance of the breast milk averaged the same, and there was no apparent difference in the rapidity with which the birth weight was regained. It seems evident that the newborn infant comes prepared for a period of non-nourishment and dehydration, awaiting the appearance of the mother's milk. In cases where the mother's milk does not appear until late, or is insufficient in quantity, the fluid requirements are taken care of by complemental feeding. As concerns renal infarctions, it is probable that these are present before birth in the large majority of cases and that the administration of water or other liquids has no effect on their prevention. It has been our practice now for sometime not to offer newborns any water whatever, unless there develops some definite indication. This is apparently true for the Minnesota climate, but may require modification in tropical or subtropical countries.

When to Use Complemental Feeding.—The question arises when should an infant be given complemental feedings, if the maternal supply is not sufficient. It is taken for granted that no food of any kind is given until a proper period for the development of the breast milk has been allowed. It has been amply shown that newborn children have sufficient reserve food to take care of their wants for several days. Several times the experiment of feeding newborns immediately after birth has been attempted to eliminate the initial weight loss. These have shown that to some degree the loss can be avoided, but at least a part of the weight loss is due to other influences than the lack of food. One must be guided in his decision as to when to give other food by a study of the child's weight curve and of the mother's milk supply As a rule newborns loose in weight for three or four days and then begin to gain. The amount lost varies considerably with the size of the infant, its state of nutrition, and various factors elsewhere discussed. At the third or fourth day the mother's milk begins to appear in considerable quantities. If by the fourth day the baby is still loosing in weight, and the mother has but little milk, we begin to consider the addition of some complement. However, it is generally better not to give additional food until a day or two more have elapsed, otherwise the child will learn to prefer the bottle feedings and nurse poorly. This removes the necessary stimulus from the breasts and weaning soon results. The plan of giving additional food was tried at the University of Minnesota and it was discovered that too early complementing frequently resulted in definite reduction of the milk supply.

Occasionally, an infant will be seen, who on the fifth or sixth day is still loosing weight and whose mother is secreting only a small amount of milk. It is then necessary to give some other food. The breast must, under no circumstances, be discontinued, but the indication is for increased activity in the attempt at breast feeding. After each nursing a sufficient quantity of complemental food is given to bring the total up to the required amount. Whenever possible in institutions or elsewhere, the complement should consist of breast milk expressed from some mother with an excessive secretion. If this is not obtainable, a simple milk mixture is the best, consisting of one-third whole cow's milk, two-thirds sterile water, and 5 per cent. of the whole of lactose. No attempt is made to imitate breast milk, most pediatrists long since having given up such efforts, but the required calories are supplied as cow's milk and sugar. Lactose is the sugar of choice in well infants. As the breast milk increases, as it almost certainly will, the complement is reduced or discontinued altogether. Frequently, only a day or two of complemental feeding is necessary. Care should be taken to give the bottle feeding after the breast nursing, so as not to lose any of the stimulating effect of nursing. The milk mixture must not be too sweet, nor too easy to obtain from the bottle, or the infant will soon prefer to neglect the difficult breast nursing and wait for the easily obtained and sweeter bottle feeding. There is nothing difficult to carry out about complemental feeding and its advantages over total weaning in cases of insufficient breast milk are so great as to leave no room for argument for the other method.

Relation of Feeding to "Inanition Temperature."—Something should be said here concerning the relationship of feeding to "inanition temperature" although the subject itself will be treated in detail in another section. This is sometimes called "dehydration temperature," but attention has already been called to the series of 350 babies in which water was given ad libitum from birth. Unexplained temperature occurred with the same frequency in these children as in 350 controls. The influence of water on their condition is, therefore, not great. Yet there can be no doubt but that the administration of food is followed by recovery. In these cases we make an exception to our general rule and give complemental feeding as soon as the fever develops to any extent.

TECHNIC OF NURSING

Position of Mother and Child.—The position of the mother and babe while nursing is a simple thing, but errors are frequently made, which have a detrimental effect upon the whole nursing program. Most of these errors are committed either through ignorance or excessive zeal on the part of the mother. Such grotesque mistakes as the mother placing the babe on its back while she kneels above it, have been described, but most of the difficulties are due to careless holding of the infant so that it tires quickly, cannot get a good hold on the nipple, or has difficulty in breathing. Some young mothers hover over the child continually in a nervous state of mind, thus distracting the babe's attention and exhausting themselves. Both mother and babe should be perfectly comfortable and relaxed. While the mother is still in bed, all that is necessary is for her to turn on her side, with her head supported, and allow the babe to lie parallel to her and nurse the dependent breast. It is well for the mother to hold her finger on the breast just above the baby's nose so that she can press the gland away from him in case he has difficulty in breathing. After the mother is able to sit up, it is best for her to sit in a low comfortable chair with her arm supported either upon her upraised knee, or the arm of the chair, holding the baby in its hollow. A footstool is often of value. All parts of the child must be supported in order that muscular relaxation may be complete and his only effort the sucking movements.

Details to Be Observed in Giving the Breast.—If a child is inclined to be fretful or nervous it is much better to have the room quiet and all its occupants composed so as not to distract his attention.

Retroflexion of the head when not properly supported leads to interference with breathing and swallowing, and consequent disturbance in nursing. It should accordingly be guarded against. It has been recommended by some writers to elevate the breast so that the nipple points upwards in cases where the breast discharges very rapidly and tends to choke the child.

Care of the Nipples.—In many cases the success or failure of breast feeding may depend upon the care given the mother's nipples. Thousands of infants lives have doubtless been lost through early weaning as a result of deformities or fissures. Many of these conditions can be prevented and practically all of them so remedied as to make nursing a distinct possibility. The care should begin during the latter stages of pregnancy. Flat or depressed nipples can frequently be drawn out by means of sucking and manipulation so that a child can grasp them. Actually inverted nipples do not give such favorable results, but an attempt to bring them out should nevertheless be made.

Before and after each nursing the nipples should be thoroughly cleaned with sterile water. Boric acid solution is commonly advised but in our belief is unnecessary. A sufficient area about the nipple must be included to prevent the child carrying any material from the surrounding parts onto the nipple. The purpose of this washing is twofold: First, it tends to prevent the infection of fissures which may be starting, and second, it prevents the child from swallowing any infectious material with his food. If the nipples are tender, the skin may be toughened by cold sponging, applying 50 to 75 per cent. alcohol several times daily, or by solutions of tannic acid. If actually cracked, more careful attention is required. Most authors advise nursing through a nipple shield. This is sometimes a valuable procedure, but so frequently the shields do not fit well, or are so constructed that the nipple is drawn into the shield and impacted there that the condition is often made worse rather than improved. A much better way is to have the milk expressed from the breast for a few days and fed to the baby in a bottle. This does not injure the nipple, nor impair the supply, and at the same time gives the lesions an opportunity to heal. The healing can be facilitated by touching up the cracks with a solution of silver nitrate. Considerable improvement is frequently noted following the application of lead nipple shields when the infant is not nursing.

Between the nursings it is well to keep the nipples covered either with a clean cloth or a piece of sterile gauze. Care should be taken, especially during the early weeks of lactation, to avoid overfilling and congestion of the breasts. If there is milk still left in the breast after the infant is through, the excess should be expressed thus avoiding stasis with its danger of reduction of the secretion.

Very heavy and pendulent breasts may be supported by an appropriate binder, provided they are a source of distress. The binder should be so constructed as to support rather than press upon the glands. Corsets and other forms of clothing which press upon the breast and interfere with its circulation are to be avoided.

Breast Pumps and Shields.—The breast pump has become progressively less used in the last few years. The objections to it are numerous. One of the greatest is its inefficiency, it being impossible to use a breast pump continually and maintain the supply at its former level. The best pump made cannot make nearly as complete an evacuation of the breast as a nursing infant or a manual expression. There is also increased danger of infection to the breast through a dirty pump, and similarly danger of contaminated milk being given to the child. The several types now in use are all constructed on the same principle. The instrument is made from glass because it is easy to keep clean. A rubber ball furnishes the suction power, alternate compressions and expressions, of which draw the milk out of the breast. The milk pours into a reservoir before it can be sucked into the rubber ball.

Many pediatrists do not use nipple shields or breast pumps at all, partly because they are a great nuisance to keep clean, but chiefly because better results can be obtained by other means.

Care of the Baby's Mouth.—Since Bednar and Epstein's studies of the infant's mouth, the care of that orifice has been much simplified. Formerly, various procedures of washing out and swabbing the mouth were practiced, but the above authors showed that most of the infections and diseased conditions of babies' mouths were due to the measures directed toward their prevention.

The care of the baby's mouth now consists in leaving it entirely alone. One cannot be too careful in supervising the cleanliness of all things that come in contact with the oral mucous membrane, but aside from keeping the exterior and particularly the corners of the mouth clean, nothing is done. A swallow of water after the feeding to rinse out the mouth may be of value, but is not necessary. However, we should not neglect to inspect the infant's mouth in order to combat any infection which might arise.

Method of Determining Amount of Breast Milk.—It is absolutely necessary for anyone who expects to carry out successful breast feeding, to accurately determine the amount of milk obtained by the nursing infant at the breast. This can be done by careful weighing immediately before and after the nursing. A good scale, graduated to measure fractions of an ounce is essential.

The infant is prepared for nursing and weighed, and when nursing is completed he is again weighed, no change having been made in his clothing. Many babies have a movement of the bowels while at breast, but changing of the diaper must be postponed until after the weighing. The weight of 1 c.c. of milk is approximately 1 gm. and the difference in the two weights in grams is accordingly the number of cubic centimeters of breast milk obtained. Of course the amount of food an infant receives varies not only with the different feedings during the day, but also varies from day to day. This variation may be as much as 200 to 300 gm. (7 to 10 oz.). Therefore, when an accurate measure of the secretion of breast milk is desired, and this is frequently very necessary to a complete understanding of the situation, the rule of Czerny and Keller to take the average of all feedings for five days, must be carried out. Physicians are particularly to be cautioned against basing any of their conclusions or treatments upon the results of a single nursing in the office or dispensary, for the unusual surroundings will often give a much lower amount than normally, aside from the error due to the variability mentioned above.

Interval between Nursings.—The determination of the length of the interval between feedings has been the cause of much discussion, among physicians, some of whom believe in a short interval, while others favor a longer period. By a long interval, we mean three or four hours. A short interval is anything less than three hours. The advocates of the short intervals believe frequent nursings give a greater stimulus to the breast, increase the breast milk in this way, and prevent over-distension of the stomach. On the other hand, those who employ the longer intervals, maintain that the breasts have more time to recuperate, and that as a result, more milk is secreted in one or two nursings than in three or four on the short schedule. The infant's appetite is keener and he is more rested and nurses with greater vigor. This seems to be born out by actual measurements. It is also pointed out that the size of the infant's stomach is not a measure of the amount of food it is able to take at a single feeding. Children in the first week have been observed to take over 200 c.c. at one time, the average capacity of the stomach probably not being over 60 to 70 c.c. This is accounted for by the fact that the excess milk passes immediately into the duodenum. Camerer[16] states that the short nursing periods cause too frequent awakenings and that the child gets insufficient sleep. He recommends five or six feedings in the 24 hours and cautions against more than eight nursings in one day at any time. Probably an infant's stomach is seldom empty within two hours after a feeding and many writers believe it injurious to give additional food to that partially digested. It must certainly slow up the process of digestion as a whole, whether it does any other injury or not. It has been suggested that when the breasts are frequently emptied the percentage of fat is increased, because of the small quantity of milk present; in many cases this is not to be desired.

The advocates of the long feeding interval have rapidly been gaining headway. We have been using the longer interval method ourselves for years, usually employing the four hour interval with but five feedings in 24 hours. The cases are very exceptional which will not do well on this plan, and the task of nursing is made lighter and more attractive to the mother, as she is thus permitted to leave her home for quite a long time The greatest advantage of the three or four hour schedule, however, is its insurance against overfeeding, the incident colic, and sequelæ of those troubles.

Number of Nursings in 24 Hours.—The number of nursings in 24 hours is of course dependent upon the frequency of the feedings. Some men have infants fed 10 to 12 times daily. As said before, Camerer states that eight is the maximum number that is safe. The following tables indicate satisfactory schedules:

	6 a.m.	6 a.m.	6 a.m.
	9 a.m.	9 a.m.	10 a.m.
	12 m.	12 m.	2 p.m.
	3 p.m.	3 p.m.	6 p.m.
	6 p.m.	6 p.m.	10 p.m.
	9 p.m.	9 p.m.	
	once at night		
Total:	7 feedings	6 feedings	5 feedings

Ordinarily we advise the third schedule, with the caution that in cases of underfeeding both breasts must be given at once as discussed below.

Regularity in Nursing.—Especial emphasis should be placed upon regularity in nursing. Many healthy babies are fed at irregular intervals, either whenever they cry, or whenever it is convenient for the mother. These babies are healthy in spite of the regime, not because of it. Their natural rugged constitution, and the nature of the milk supply protecting them. Many others who are irregularly nursed come to grief through overfeeding, with its accompanying symptoms, which often result in weaning. It is very important that even a newborn child should acquire regular habits, and have certain times allotted for eating, resting, and playing. Such habits are an aid to digestion and insure a more uniform breast milk, both in quantity and quality. If a normal infant is started from birth on a regular schedule which is rigidly adhered to, he will as a rule awaken with great punctuality just a short time before the feeding. No other factor is more important in stabilizing the child's nervous system, and incidently the peace of the household.

Rotation of Breasts.—In cases where there is a sufficient supply of breast milk and the child is strong and nurses well, it is usually best to give only one breast at a feeding. At each feeding the alternate breasts are given so that each one is only nursed once in eight hours. This protects against overfeeding (but it must also be ascertained that the baby is getting enough), provides for complete evacuation of the breast at each nursing, and insures a more uniform quality of milk. If a child is not getting enough food in this way, it is frequently necessary to give both breasts at each feeding. When this is done it is well to alternate the breast to be given first at each nursing. The infant in this way obtains practically all the milk present in both breasts, and also gives each breast a complete evacuation every eight hours, thus stimulating the milk production. This latter plan is quite safe and usually leads to an increased secretion but when both breasts are given at shorter intervals, one's purpose is often defeated and the milk supply decreases, owing to the failure of complete evacuation. If the supply of milk is abundant and the child premature or very weak, it may be better to give both breasts for a short time so as to save the infant's energy, but this should never be done to an extent which would jeopardize the secretion.

Duration of a Single Feeding.—The duration of a single nursing depends to a considerable extent, upon the character of the breast and the vigor and appetite of the child. Breasts which yield their milk rapidly will be emptied in a much shorter time than those which yield only with difficulty. One child may empty a breast in half the time it will take another and less vigorous infant to empty the same breast. However, it has been fairly well established that there is little use of keeping a child at one breast longer than 20 minutes. Weighing at frequent intervals during the nursing has repeatedly shown that most of the milk is obtained within 5 or 10 minutes.

The figures given by Feer are typical. An infant observed by him nursed 112 gm. in the first five minutes, 64 gm. in the next five minutes, and only 10 gm. in the third five minutes. A statement frequently quoted states that more than half of the total is received in the first five minutes, more than half of the remainder in the next five minutes, and the remainder thereafter. If a child nurses vigorously for half an hour or longer, it is an almost positive sign of insufficient breast milk. Occasionally, a child that is very weak may have to rest at intervals during the nursing, and thus require a half hour or more, but it is very questionable whether such a child should go to breast, but rather should be fed the expressed milk either from a bottle or by tube.

Amount Obtained at Breast.—While a considerable individual variation exists in the amounts of breast milk taken, yet the average of different groups is quite similar. That the size of the stomach does not control the capacity of the infant has been shown by Scammon's [17] figures. After the fourth day infants uniformly take at each nursing an amount in excess of the anatomic capacity of the stomach. Infants in the first week have repeatedly been observed to take between 200 to 250 c.c. at a feeding when the anatomic capacity was not over 60 to 70 c.c. The amounts taken in the first few days are dependent wholly upon the ability of the breast to secrete, infants being able to nurse vigorously soon after birth. Camerer gives the following table for the first week which is approximately the same as that observed by most physicians.

Day	Amount, cubic centimeters
1	10–20
2	90
3	190
4	310
5	350
6	390
7	470

Considerable variation between individual nursing during the day and even successive days is noticed, but Scammon, in analyzing and averaging a large number of feedings in the University Hospital, has shown that no constant relationships are observed. All these children were on four-hour schedule and the morning feedings averaged no higher than any other. This might not be true if a shorter interval were employed since after four hours the breast seems to reach its maximum secretion, a longer wait not increasing the amount. The following table by Grulee gives the approximate daily quantities obtained during the first six months.

Time	Amount, cubic centimeters
1st week	250–300
1st month	600–650
2nd month	815–870
3rd month	800–850
4th month	850–900
5th month	900–950
6th month	1000

Giving Water between Feedings.—Something has been said before, concerning the administration of water between feedings. Czerny and Keller do not believe it is necessary to give any at all, maintaining that the milk contains sufficient fluid. It is also true that giving water can do little harm except to disturb the child by awakening him and causing more frequent urination. If given within an hour before nursing it is possible that it may impair the appetite. Camerer gives water during the first week only when the secretion of breast milk is small and thinks that by so doing he prevents the formation and collection of uric acid in the kidneys. Ordinarily, it is unnecessary to give an infant nursed at a properly functionating breast any other fluid aside from the milk supplied by the mother.

MIXED FEEDING

Indications for Mixed Feeding.—Mixed feeding, that is, the employment of some other food with the breast milk, is indicated when the supply of mother's milk is insufficient for the nutrition and growth of the child. There is absolutely no question about the advisability of this form of feeding under the above circumstances. Total weaning should not take place simply because the supply is inadequate. Objection has been made to the use of cow's milk and breast milk together, but such objections are entirely unfounded. In case of subsequent alimentary disturbances, the small amount of breast milk is of invaluable assistance in their control. The increased resistance and aided digestion resulting from relatively small amounts of breast milk is of inestimable value.

Complemental vs. Supplemental Feeding.—Supplemental feeding consists in the substitution of a regular nursing by a bottle feeding. Complemental feeding consists in giving some additional food in a bottle immediately following a regular nursing. The former is used in cases of insufficient breast supply when a short interval is used. It is also used when weaning is desired with the long interval plan. Complemental feeding is used when the breast supply is insufficient but when we desire to maintain the supply at its highest possible level. Complemental feeding is greatly to be preferred to supplemental feeding, purely because of the influence on the breast secretion.

When entire nursings are omitted and the breast is not stimulated for long periods, the already small secretion will rapidly decrease. The amount of the complement must, of course, be determined by the amount of breast milk obtained. The complemental food is then added in sufficient quantities to supply the necessary calories to the child, or until he is gaining satisfactorily.

Foods Used in Complemental Feeding.—The foods used in complemental feeding are the same as those employed in total artificial feeding, preferably simple milk mixtures containing lactose, the strength of which varies according to the age of the child, as elsewhere described. Some observers state that infants complemented with protein milk do not do well. We have never noticed this to be true. Fortunately, protein milk is seldom indicated when some breast milk is being received.

PATHOLOGY OF BREAST FEEDING

Introduction.—Nutritional disturbances in breast fed infants are very frequent, but fortunately are usually so mild that they are either entirely unnoticed by the parents or do not require the consultation of a physician. Infants are fitted to digest human milk better than any modification of any other milk. Nutritional disturbances, therefore, seldom indicate fault with the maternal food but are usually due to over- or under-feeding, acute or constitutional disease in the infant, or faulty habits of hygiene and nursing. Children vary widely in their abilities to digest even breast milk, but when, under rare conditions, a child does not tolerate breast milk well, there is usually no other food which will do as well and treatment must consist in modification of the breast milk or the substitution of a different nurse. Most of the feeding troubles of breast fed infants correct themselves but occasionally become chronic and serious either at that time or at the time or weaning.

Contra-indications to Breast Feeding

Every physician, whether pediatrist or not, should be conversant with the contra-indications, imaginary or real, to breast feeding, because he may at any time be called upon in one capacity or another, to help decide for or against this important proceeding. The responsibility is very great. Upon his decision may depend the life of the infant. The question is not always an easy one to decide, and often, after careful consideration, subsequent events will show that the wrong course has been followed. One essential principle should be kept in mind always, whenever the issue is not clearly defined, always decide against weaning. Enough has already been said concerning the value of breast milk to convince everyone of the absolute necessity of preserving its secretion whenever possible. According to our experience, all of the contra-indications to nursing, except one, are a matter of specific decision in the individual case.

Disease in the Mother. *Tuberculosis.*—This constitutes the only positive contra-indication to breast feeding, and that when the tuberculosis is active. It is evident that a mother with open pulmonary or laryngeal tuberculosis cannot nurse her child, either in justice to the child or herself. It is probably too dangerous to attempt to nurse when the active disease is located in the joints. On the other hand, from the infant's standpoint, when the lesions are healed, whether in the lungs, joints or elsewhere, there is no objection to nursing. Danger of infection in these cases is for the most part past, and the only consideration here is the strength of the mother and the liklihood of the burden of nursing causing a recurrence of the trouble. In most cases this latter should be determined by trial and careful supervision, many very delicate women improving their general condition while nursing. However, the most careful supervision must be maintained and frequent examinations for active trouble made, in justice to both the parent and child.

Epilepsy and Insanity.—These are ordinarily given as definite contra-indications to nursing but in our opinion this is purely a relative matter. It is hard to conceive that any factor can enter into the question aside from the possibility of the mother injuring the child while mentally unbalanced or during a convulsion. There certainly is no physiologic reason why milk secreted by an epileptic or insane woman is not just as fit food as that secreted by any other woman. If the mother's condition is mild or if the circumstances are such that the infant can be watched by some responsible person, nursing may be carried on just as satisfactorily as with a normal mother. Certainly the dangers of harm coming to the child are much less than when a newborn baby is placed immediately upon artificial food.

Nephritis, Puerperal Convulsions, Anemia, Postpartum Hemorrhage, Cardiac Disease, Etc.—These conditions may alter the qualities and quantities of the breast milk to some extent, and it is conceivable that in a severe case of nephritis or eclampsia that considerable amounts of nitrogenous or toxic substances might be excreted in the milk. Usually in cases of eclampsia it is advisable to discard the breast milk for a few days. However, these effects would probably never be sufficiently marked to require permanent discontinuance of the breast. The reason why these conditions not infrequently necessitate weaning is not through their effects upon the child but upon the mother. Anemia, and postpartum hemorrhage are usually only temporary conditions and recovery is usually sufficient in a week or 10 days to permit resumption of nursing without danger to the mother. In the other conditions it is often incompatable with the mother's condition to attempt nursing at all, but fortunately, women of this type are seldom able to bear children and we accordingly meet the problem but seldom.

Malignant Disease.—Severe malignant disease in the mother, particularly when involving the mammary glands sometimes precludes nursing both because of the mother's physical and psychical states.

Such disease in no way influences the quality of the mother's milk and as long as she is possibly able the nursing should be continued. In the slowly progressing types, located away from the breasts, there is absolutely no reason why breast feeding cannot be carried out successfully.

Syphilis.—Syphilis in the child or mother is not a contra-indication to nursing unless the disease has been acquired since the child's birth. It has been amply shown that otherwise both are already infected. Moreover, if we expect to treat the child's disease successfully it is very important that he have breast milk, since children with congenital syphilis are especially prone to nutritional disorders and tolerate artificial food very badly. It stands to reason that a syphilitic infant should never nurse anyone but its own mother and vice versa.

Acute Infectious Diseases.—Most authors recommend the temporary weaning of the babe during the acute infectious diseases, but put him back to the breast in a short time. It is usually necessary in such severe diseases as pneumonia and typhoid fever to remove the child from the breast so as to conserve the strength of the mother as much as possible, but from the standpoint of the infant there is no reason why nursing should not be continued, except for the lessened supply. It is our custom not to remove the infant during the other infectious diseases unless for some specific indication. Young infants are immune to certain of the exanthemata and can be definitely immunized to diphtheria and small-pox. Competent aseptic nursing is all that is necessary to prevent the transmission of other diseases from the mother to the babe, it being fairly well established that the milk does not transmit these diseases.

Pregnancy.—It is a frequent experience of every pediatrist to encounter children with grave nutritional disturbances who, we learn from the history, have been weaned because the mother is again pregnant. It has been shown by Eckles that the supply and composition of the milk of cattle is not influenced by pregnancy during the first half of the lactating period, but that after that there may or may not be a reduction of quantity dependent upon the adequacy of the diet. No observations have ever been made on pregnant nursing women of a very accurate character but daily experience is sufficient to convince one of the fallacy of weaning because of pregnancy. Cases where a woman nurses one child up to the birth of the next are not uncommon and those that nurse up to the sixth or seventh month of pregnancy are frequent. Among certain of the lower classes where babies are borne with great frequency, lactation is almost a continuous state with many of the mothers. Probably the origin of the idea that pregnancy is not compatible with nursing depends upon the frequent reduction of the supply after the mother learns her condition. This is in turn dependent upon the nervous shock which she experiences and too insufficient or inadequate food intake. It is, therefore, of the utmost importance to maintain the woman's nutrition at the proper level and put her mind at rest. If these things are done all babies can be nursed until at least their sixth or seventh month even in spite of the mother's pregnancy.

Menstruation.—A little disturbance in the comfort of the infant, a slight irregularity or looseness in his bowels, or perhaps a little vomiting is sometimes noted during the mother's menstrual periods and has erroneously been regarded as sufficient cause for weaning. Such manifestations are extremely inconstant and by no means certainly due to the mother's condition. No doubt the symptoms so frequently credited to menstruation in the mother are more frequently due to slight acute infections or temporary overfeeding. However, there are cases which show this phenomena with great regularity at the mother's period, but they are never severe and seldom more than a day or two in duration, and never a cause for discontinuance of the breast.

Inability to Nurse Previous Children.—Recently one author has given as a contra-indication to nursing the inability to nurse the previous two children while under careful supervision. Such a statement is ridiculous. No matter how good the supervision may have been no physician is so certain of himself that he might not have overlooked conditions which now are removed. We have seen mothers who had tried sincerely to nurse three or four previous children without favorable result, but who subsequently were able to nurse as well as any woman. In any case the child should be given the benefit of the doubt and the attempt at breast feeding continued until the inability is manifest. We believe that if the supervision of the nursing has been of the proper kind that no such situation can result as all women will secrete some milk at least for a short time after childbirth.

The same author gives as another contra-indication to breast feeding, prolonged gastric or intestinal disease in the infant. The proposition here is, that if a breast-fed infant has been having gastric or intestinal disturbance for a long time in spite of careful regulation of feeding, that artificial food should be employed. According to our conception of infant feeding prolonged gastric or intestinal disturbance is the paramount indication for breast feeding, whether the previous diet has been artificial food or mother's milk. Under circumstances such as those described above, the physician has failed to recognize some fault in the breast feeding, usually in regard to quantity. That does not mean that he therefore should stop the maternal feeding and institute some form of feeding about which he knows even less, but that every attempt should be made to locate the fault and correct it. In case the fault cannot be located the physician will do better to continue breast feeding than he will to substitute an artificial food which he cannot know the infant will tolerate. It is axiomatic that a child that cannot tolerate breast milk, the most easily tolerated of all foods, cannot tolerate any other food as well. We could think of no greater folly than weaning a child for the above causes.

Summary.—In general, then, there is but one absolute contraindication to breast feeding: active tuberculosis in the mother. All other indications are relative, depending upon their degree of severity, the physical condition of the mother, and the environment. In all these cases it is the physician's individual responsibility to decide for or against nursing, and, as has been said before, the responsibility is a very great one. The wise physician will always give the benefit of any doubt to continued breast feeding. Unfortunately, many, not knowing what to do, wean a child and make the situation worse.

OBSTACLES TO BREAST FEEDING

By an obstacle we understand some factor which renders breast feeding difficult, but which does not preclude its conduct. Such factors may be located either in the child or in the mother or both. Their presence is often a source of great tribulation to the pediatrist but the satisfaction of overcoming them and the benefit derived by the child more than compensate for the difficulties.

On the Part of the Child. *Malformations.* (1) *Hare-lip.*—This constitutes the most frequent of the malformations that interfere with breast feeding. If the palate and gums are intact, however, there is little interference with nursing during the first few months because the act of nursing is carried on by the pressure of the nipple against the hard palate by the lower jaw, the lips really taking but little part in the act. By the time the lips begin to be of importance in nursing, an operation should have been performed and the lip healed. We have operated in mild degrees of hare-lip within 24 hours of birth. The operation should be done as soon as the child's strength will permit, which is usually very soon.

2. *Cleft-palate.*—This offers much greater difficulties to nursing than does hare-lip. A great deal depends upon the degree of the deformity. If only the soft or posterior part of the palate is involved, little interference is to be expected, but when the cleft is complete or, worse still, bilateral and involving the alveolar arch, great difficulty may be encountered. This is purely a mechanical proposition as can be readily understood from considering the mechanism of nursing. If the palate offers sufficient resistance to the nipple as it is pressed upwards by the mandible, the milk may be expressed fairly readily and the only difficulty will be that the child must interrupt the nursing every few seconds in order to breath. Some milk may be lost through the nose but not to any considerable degree. In the more severe grades nursing is often impossible, but breast feeding should be carried out at all costs, for the little patient has ahead of him a strenuous operation during which he will need every possible degree of strength and resistance. The breast milk should be expressed from the mother's breasts and fed to the child either with a spoon, a medicine dropper, or by stomach tube depending upon the degree of malformation. As soon as possible the operation should be performed. Brophy, who

probably has the greatest experience, is an ardent advocate of early operation.

Feeble Nursing.—Occasionally a mother will have good breasts, good nipples, and abundant milk supply but the infant does not nurse at all or so feebly that he obtains insufficient food. Usually, there is some definite reason for this, either the child is premature or there is present some constitutional disease or defect. The possibility of cerebral hemorrhage must always be considered. Certain infants who are apparently normal are also occasionally affected in this way, but the condition is usually temporary and soon passes over. In all these cases, no matter what the cause, the important thing is not to allow the breast secretion to diminish. At first every attempt should be made to improve the child's ability to nurse and every possible obstacle removed, and then the breasts should be evacuated mechanically and an appropriate amount given to the child. A bottle may be used to give the food or perhaps it may be necessary to feed by gavage. This obstacle is a trivial one and should give but little trouble.

"Fear of the Breast."—Although the author has never personally seen such a case, authentic reports have been made of children who refuse to nurse, turn their head away, cry lustily, and appear to be afraid of the breast. When such infants are blindfolded or nursed in a dark room it is said that they take the breast well and nurse like any other child. Whether or not this is truly a condition of fear in the infant or a habit based upon a neuropathic diathesis, formed by improper technique in nursing during the first days of life, is a matter of considerable doubt.

On the Part of the Mother.—The question of poorly developed breasts, malformations of the nipples, and fissures of the nipples has been taken up in the preceding chapter. It will be remembered that these obstacles can be overcome in a number of ways, by the use of nipple shields, by breast pumps, but best of all by manual expression of the affected breast. Mastitis is a frequently encountered obstacle which greatly concerns the attending physician and often leads him to discontinue nursing. Mastitis, even in the presence of extensive pus formation, involves only the stroma of the mammary gland and is exclusively an interlobular affair, involving the parenchyma only by erosion. In the earlier stages of mastitis and even where an abscess has been opened and is draining externally, there is no reason why the child should not continue to nurse. Most of these cases do not show any abnormalities in the milk, and even the presence of a few pus cells does no harm. Care of course should be taken to cleanse the nipple and surrounding areas. Only in the presence of a discharging abscess in the areola or when an abscess has ruptured into one on the larger milk ducts should discontinuance of nursing be advised and then only temporarily on the affected breast. As a matter of fact the continued nursing is of benefit to the diseased breast as it avoids engorgement and consequent tendency to spread of infection. Therefore, we seldom discontinue nursing because of mastitis.

A condition known as hypogalactia, or slow secretion of milk, is described by some writers. There is much doubt whether such a condition ever exists. The construction of some nipples prevents the rapid discharge of the milk, and many breasts may have only small amounts, but we have never been convinced that an otherwise normal breast will give up its secretion so slowly that the infant cannot obtain a sufficient amount.

UNDERFEEDING

With most of the infants who are brought to the physician because of digestive disturbances, the mother and relatives think the child is not getting enough to eat. Usually their only foundation for this idea is that the child is fretful, cries considerably, or sucks its fists, symptoms not necessarily related to underfeeding. Seldom are these infants underfed but suffer from some other alimentary disturbance. Frequently, on the other hand, children who are much underfed are not suspected of it by their attendants.

Etiology.—Obviously the etiology of underfeeding must result either from an insufficient quantity or quality of breast milk. Probably the matter of quality very seldom if ever is the sole cause of underfeeding. The protein and carbohydrate constituents of breast milk are fairly constant in all samples, the fat being the only constituent which varies widely. Even milks poor in fat contain sufficient amounts for the nutrition of the infant when furnished in the ordinary amounts. Rarely is breast milk encountered, which, when fed in proper amounts will not sufficiently nourish the infant. By far the most common cause of underfeeding is insufficient quantity. This may depend upon several factors. According to Birk, about 13 per cent. of mothers nursing children for the first time have an insufficient quantity throughout the nursing period. Others who have abundant supplies at first not infrequently suffer rather sudden reductions of quantity without their being aware of it. It is stated that the cause of underfeeding is sometimes to be found in a breast which yields the milk with great difficulty, so that even though the supply is abundant, the infant becomes so fatigued that he stops before obtaining the full amount. In these cases another element is often added in that the mother suspects the condition and puts the child to the breast more frequently. Disaster often follows for now the milk flows no better and the infant's appetite is not so keen and he nurses even more poorly. Late appearance of the breast milk really is not a causative factor in underfeeding but results in an inanition during the first few days of life, which should be cared for by complementary feeding and which will disappear when the breast milk comes in.

Symptoms.—The symptoms of underfeeding are dependent to a large extent upon the degree of the trouble. In the very mildest cases nothing at all may be noticed except that the baby does not gain as fast as normally and may be a pound or two underweight at the fifth or sixth month. In all cases the first symptom is stationary weight which is soon followed by a more or less rapid loss of weight. Contrary to the impression held by mothers and their advisors, underfed babies do not necessarily cry a great deal. Occasionally their sleep is restless but, for the most part, fully as sound as that of a normal baby, and when the condition is advanced, they sleep much more than normally, indeed often almost continuously. This is probably one of Nature's methods of conserving all the available energy of the child's body. Underfed babies do not vomit, which is an extremely important point in the diagnosis. The stools are either much smaller than normal or much more infrequent. Often the physician first sees the child because of this pseudo-constipation. They are not the large hard stools seen in dyspeptic infants but conform to the so-called "hunger" type, being small, dark colored, pasty, and completely digested. Mothers frequently notice that urination is scanty and that the diaper needs changing but seldom. The urine becomes very concentrated and will often leave a stain upon the diaper and have a strong odor. It is typical for these babies to remain at the breast for a long time, even an hour or more. Often they evidence their dissatisfaction by restlessness. They will release the nipple for a moment but immediately take it again. As a rule they nurse vigorously and continue until they are exhausted and stop from shear fatigue. Not infrequently they fall asleep on the breast. As the ailment progresses they become weak and apathetic. The skin is pale and its turgor distinctly reduced. The temperature is usually subnormal, sometimes several degrees. Anemia is progressive. The abdomen is sunken and the fontanelles depressed in the extreme cases, and the whole appearance of the child is pathetic. In breast fed children the severe degrees of undernourishment are seldom seen because the condition becomes manifest and some means is taken to overcome it.

Diagnosis.—The diagnosis is usually evident after obtaining the feeding history and making a physical examination. However, one must rule out dyspepsia, but this seldom is difficult because of the marked gastro-intestinal symptoms in the latter. Underfeeding is characterized by a falling weight curve without gastro-intestinal symptoms. This, taken with a history of frequent or prolonged nursing, is usually sufficient. However, a positive diagnosis can be made only by actual measurement of the food intake, by weighing before and after feeding. Here, again one must be warned against drawing conclusions from a single nursing. Reliable information can be obtained only after 24 hours and better after four or five days.

Treatment.—The treatment of underfeeding at the breast is to supply more food. This may be done through regulation of nursing, stimulation of the breasts, or complemental feeding. A frequent error among the advisor's of many mothers at this time is to conclude that the milk is not agreeing with the infant and instruct the mother to wean the infant immediately. A still more frequent error, and less forgivable, is to wean the infant at this stage because the supply is insufficient, and not to consider complemental feeding. Under no circumstances is weaning to be considered. The first thing to do in each case is to stimulate the breast to secrete the maximum amount of milk of which it is capable. First the habits of mother and child must be carefully regulated. The child must not be nursed so frequently, nor left at the breast so long that its strength is exhausted. Likewise the mother must be guarded against fatigue and nervous strain. The peace of mind of the mother together with plenty of food and rest are the first essentials to increasing the supply of milk. Aside from ascertaining that the food intake is ample and that a little extra fluid is taken, we seldom make any dietary recommendations.

The first step in nearly all cases is to give both breasts instead of only one at each nursing. This gives an added stimulation from the child itself. Care must be taken, however, to alternate the breast to be given first so as to provide equal stimulation for both. The infant's nursing time is not lengthened but divided between the two breasts. Usually we recommend 12 to 15 minutes at each breast. Some shorten the interval between the feedings, but in our experience this is rarely necessary and seldom advisable. The supply of milk is then little if any increased for the 24-hour period and the child is in great danger of becoming fatigued so that he nurses more poorly than before and our own purposes are defeated. It may sometimes be well to add another nursing during the night but it seldom helps to shorten the interval.

Such measures as massage, Faradization, and steaming of the breasts have proved to be of no value in our experience. Bier's hyperemia, so highly praised by some, is likewise ineffectual. All that such measures can accomplish at best is an increase of blood supply to the gland, which as far as we know has no particular physiologic relationship to the production of milk. Galactagogues are of no practical value, pituitrin being the only one which has any demonstrated effect, and that for a period of only about 10 minutes following its injection.

However, of extreme value and easy performance is the stimulation provided by complete evacuation of the breasts at each nursing. It is a fundamental physiologic principle that the function of any organ is directly in proportion to the demand made upon it up to a certain limit. Accordingly if after each nursing the last remaining drops of secretion are regularly removed by mechanical means the secretion very quickly increases. (The technique of this procedure is elsewhere considered.) This method is especially striking in those cases where the infant has been nursing poorly or when complemental feeding has been used. In all cases some increase in milk supply is thus possible and in the greater proportion, sufficient increase takes place to completely supply the infant. In those cases where the supply is far too small, and where after stimulation the supply is still inadequate, complemental feeding must be resorted to. Complemental feeding is always indicated in these cases and supplemental feeding never used as it provides for too long an interval in which the breasts receive no stimulation. The food to be used is the same as if the child were artificially fed, and usually consists of a simple milk mixture. The amount of complement can be accurately determined only by ascertaining the amount of breast milk and adding sufficient complement to supply the caloric requirements. An estimate can often be made, however, and a very satisfactory result obtained, by regulating the amount according to the progress made by the child.

OVERFEEDING

Overfeeding at the breast is no doubt very frequent, but Nature either provides its own remedy through regurgitation of a portion of the food or the symptoms are very mild and do not lead to complications which bring the infant to the physician. Most healthy babies probably take an excess of breast milk which they either spit up or deposit as excess fat. Usually we do not see simple cases of overfeeding except in children who are kept under observation as a preventive measure or in infants who come to us for other reasons. It is not until some complication has developed that we see most of these cases.

Etiology.—The cause of overfeeding in the majority of cases is too frequent nursing in the presence of abundant milk supply. Severe cases of overfeeding and dyspepsia are almost never seen on the four-hour feeding schedule, but do occur when feeding is irregular and carried out every one or two hours. In these cases the symptoms of "colic" and crying frequently lead to increased frequency of nursing which quickly adds to the trouble and soon leads to complications Occasionally, a child fed on a proper feeding schedule will be overfed because of an excess of secretion by the breast, but this is rare. This further is favored by a long interval for the capacity of the child is nearly reached normally and does not permit of very great excess intake. The overfeeding may be caused by an excess amount of fat in the breast milk. The symptoms thought to be attributable to too much fat may be due to reduced fat tolerance on the part of the infant.

Symptoms.—In the early stages of overfeeding the chief symptoms are a slight tendency to regurgitate part of the food, soon after nursing, and a somewhat increased frequency of the bowel movements. Occasionally the appetite will be a little impaired but that does not usually happen until later. As the condition advances the vomiting becomes more marked and is persistent. It occurs not only after nursing but in the interval as well. At first the stools become diarrheal in character, being loose and containing white soap particles (curds), but later constipation may occur as the early stages of dyspepsia appear. The most pronounced symptoms and the ones for which the parents seek relief are the attacks of "colic" and abdominal distress. These are protracted and severe, usually occurring from one to three hours after nursing, and are accompanied by considerable vomiting and abdominal distension. Visible intestinal peristalsis is sometimes present. The crying is shrill and gives evidence of the pain suffered by the infant. The child becomes exceedingly irritable and sleep is much disturbed. If the condition is allowed to continue, the weight soon becomes stationary and as dyspepsia develops, falls rapidly.

Diagnosis.—There are no difficulties presented in the diagnosis of overfeeding in most cases. The gastro-intestinal symptoms described above are very typical and when occurring in the absence of other diseases are practically diagnostic. A weight curve which has been rapidly rising before the onset of the symptoms and which becomes quickly stationary or begins to fall is also typical. The absolute diagnosis, however, can only be made by the actual measurement of the food intake in the 24 to 48 hour period. This should be done in every case where it is possible, both as a diagnostic procedure and as an index to treatment.

Treatment.—Probably no other condition is responsible for so much inadvisable weaning as is overfeeding. It is so logical to conclude that the breast milk does not agree with the child because he vomits, has colic, and diarrhea. Accordingly, many of these babies are immediately put upon the bottle because of some supposed incompatibility between the milk and the child. This error is still widely made by physicians and no doubt causes the death of hundreds of babies in this country every year. Overfeeding is never an indication for weaning.

The treatment consists in reducing the food to the proper amount by controlling the nursings. The adoption of the four-hour schedule from birth will prevent most cases of overfeeding, and should be at once instituted when a case is encountered. Perhaps the mother may have been giving both breasts at each feeding, and if so, should be instructed to give but one. If this procedure does not sufficiently reduce the quantity, the length of time at the breast must be reduced. The extent of the reduction must be determined by trial. In some cases an infant will receive more than the necessary amount in two minutes, if the supply is abundant, the nipple yields the milk easily, and the infant is vigorous. In the cases where the milk is exceptionally high in its fat content and the child does not seem to tolerate fat very well, it may be wise to have the infant nurse on both breasts for just a few minutes each. The fat comes mostly in the later portions of milk and the total amount of fat can be much reduced in this way. If this is practiced the mother's breast should be evacuated after the infant is taken from the breast so that no reduction in the milk supply occurs. These methods will control any case of overfeeding if properly carried out.

For the treatment of abdominal distress (colic) and vomiting in the acute stages other methods may be necessary. If the vomiting is severe, a period of starvation during which only water or barley water is given lasting from 12 to 14 hours is usually efficacious. Gastric lavage is also helpful. Bismuth is sometimes given but is of questionable value. The paramount object in the treatment of the distress is the reduction of food. Stomach washing and external heat help to allay the acute pain. The resort to sedatives like chloral or opium is rarely necessary.

Prognosis.—As a rule the prognosis in cases of simple overfeeding is excellent. The younger the child the more difficult, however, is the treatment because of their lower tolerance. But under proper care all cases should clear up in a few days.

Disturbance of Balance

This condition is extremely rare in breast-fed infants, or rather is so short in duration that it escapes observation. It is to be considered as a complication of overfeeding and occurs as a transition stage between simple overfeeding and the more severe complication of dyspepsia. Its cause, diagnosis and treatment is that of overfeeding.

Dyspepsia

This constitutes the most severe complication of overfeeding and is also the most severe alimentary disturbance with which breast-fed infants are frequently afflicted.

The sole cause of dyspepsia in the breast-fed is overfeeding, which may result, as before mentioned, from too large a quantity at each feeding, too high a fat content of the mother's milk, or abnormally low tolerance of the infant to any kind of food.

Symptoms.—The early history is one of overfeeding and the early symptoms are the same as for that trouble. A period of rapid gain usually precedes the onset of vomiting and distress. The weight curve is very typical in dyspepsia. A rapid rise is followed by a short stationary period which in turn is followed by a more or less rapid fall, depending upon the severity of the trouble. The vomiting and distress are marked. Abdominal distension and discomfort is frequently present. Between the attacks of vomiting considerable gas may be belched out. The stools are increased to 8 or 10 or more rather watery evacuations which are expelled from the anus forcefully together with considerable amounts of gas. The stools are usually sour and contain white and green particles. The irritating stools cause the buttocks to become red and inflamed and set up a marked intertrigo in the ano-genital region. There is irregularity of temperature. Loss of appetite becomes marked after a short time and tends to correct the disorder if other means are not employed. Loss of weight is sometimes very severe.

Treatment.—This is directed at the underlying cause and is the same as for overfeeding. Reduction of food intake below the tolerance of the infant is here the important factor in treatment. If the case is a protracted one, and much weight has been lost, and the child's tolerance greatly reduced, the reduction of food may have to be very great. The subsequent increases may have to be made very slowly and by small steps. However, the severe reduction of tolerance that occur in artificially fed infants are not seen in those fed at the breast only.

DECOMPOSITION AND INTOXICATION

These are so rare in the breast-fed infant as to hardly be worthy of mention, but they may result if a bad case of dyspepsia is neglected. They differ in no wise from the same conditions described under artificial feeding, except that they are usually much more serious and difficult to treat for now the baby has even lost its tolerance for breast milk.

FEEDING IN CONGENITAL DEBILITY

In congenital debility the infant's tolerance for breast milk is reduced at the time of birth because of some physical defect or inherited disease. Feeding in this type of child, therefore, becomes a very difficult matter and unless handled with the utmost skill, will frequently be the direct cause of the infant's death.

Etiology.—Prematurity is one of the commonest causes of reduced food tolerance in the newborn. In these little beings, the digestive functions as well as the other functions are not completely developed, and they are able to handle the breast milk only in suitable amounts. Tuberculosis, syphilis, and alcoholism in the parents are very important factors in the production of this condition. This is especially true of syphilis, in which condition breast feeding properly supervised is extremely important. Congenital malformations such as cleft palate, etc., are additional factors in producing reduced tolerance.

Symptoms.—The symptoms will, of course, depend upon the underlying causes and vary widely in the different cases. The feeding symptoms are chiefly divided into those of underfeeding and overfeeding, together with the complications of each. Often the development of nutritional disturbances in an apparently normal child that is being fed rationally will lead to the discovery of some underlying constitutional defect.

Treatment.—The treatment consists of treating the underlying disease, and in addition regulating the feeding. Feeding premature infants is considered elsewhere in this work, but the success or failure of feeding in these conditions depends upon keeping the food intake below the infant's tolerance and yet supplying sufficient food for growth.

It is extremely important that the supply of breast milk be maintained in the mother by means of manual expression, since as the child is not nursing the breasts will soon dry up.

FEEDING IN ENTERAL AND PARENTERAL INFECTIONS

When an infant who has been progressing ideally is attacked by some acute infection, no matter how mild it may be, there is an accompanying reduction of its tolerance to normal food. Depending upon the severity of the infection and the resistance of the infant the reduction may or may not be great enough to fall below the requirements of the child. Fortunately in these infections the child very early suffers a more or less marked loss of appetite which automatically tends to correct the discrepancy. It is only after severe and prolonged infections that sufficient reduction of tolerance results to produce an alimentary disturbance in an exclusively breast-fed child. These infections may be located away from the intestinal tract, for example, in the ears, throat, nose, lungs, or kidneys, or they may be located in the intestine itself.

The symptoms, aside from the elevation of temperature and local symptoms at the site of infection, usually consist of a more or less severe diarrhea associated with vomiting. Loss of weight may be very marked and abrupt. Abdominal discomfort is not infrequently present.

Feeding regulation is not necessary in the majority of breast-fed infants suffering from these infections. If any complemental food is being used it must be withdrawn at the first elevation of temperature, and breast milk only be given until conditions are again normal. Nursing should be continued on the same schedule as before. Excuse for changing the regularity of the feeding must not be found in the child's illness. If the fever is marked it will be necessary to give additional fluids in the form of water or cereal water. It is seldom necessary to reduce the amount of breast milk if the schedule is adhered to, but this may occasionally be indicated in very sick children or in children of low vitality whose food tolerance has become greatly reduced.

In general, one needs to feel little concern about the feeding of nursing babies during parenteral infections. They are, however, great contributors to infant mortality in the artificially nourished.

"Idiosyncrasy Towards Mother's Milk"

Numerous authors describe a condition which they refer to as "idiosyncrasy towards mother's milk." This is supposed to occur when an apparently normal baby vomits, loses weight, or has diarrhea while nursing a mother who has an apparently appropriate supply of normal breast milk. These babies are said to recover from their symptoms and progress normally when placed upon other food.

There is great doubt in our minds as to the existence of such a condition. We have never seen a case in which the mother's milk did not agree with the child better than any artificial food that could be found. This is also the experience of many other physicians. It is more than likely that under circumstances such as those described above some disease or defect in the infant, some fault in the hygiene of mother or child, or some imperfection in the nursing technique is at the bottom of the trouble. One cannot absolutely say that such a condition does not occur but it is a great deal more difficult to prove that it does. The diagnosis can be made only after the exclusion of every other possibility. We should certainly never recommend the substitution of artificial food in a case of this kind. We do not attach any importance to this condition, except in so far as its supposed existence may lead to inadvisable weaning.

Restitution of Breast Feeding and Increase in Breast Supply

The situation frequently arises in which we wish to restore the breast secretion or get more milk. Often the mother's supply is not adequate for the nourishment of the babe. After the mother has been ill and we have been forced to remove the child from her breasts or after a babe has been inadvisably weaned, restitution of the breast milk is many times necessary. The method of bringing about these changes is a matter of great importance.

The question naturally arises as to how long after discontinuance of the breast, restitution should be attempted. A recollection of the physiology of lactation and the theories of milk secretion will help us in deciding this. After a woman has been pregnant once, the breast is mature and thereafter not much different from the secreting gland. Attention has been called to the view of certain writers that the normal condition of the mammary gland is lactation, disappearance of which results from a lack of demand upon the organ. If, as seems possible, the corpus luteum is of influence in milk production, attempt to restore the secretion would be justifiable for four or five months at least after delivery, since we know that the corpus luteum persists that long. However, by far the most important of the determining factors is our knowledge of the influence of sucking upon milk production. We have seen that milk secretion may be started at almost any time by the process of sucking. From our clinical experience we are inclined to attempt restoration after as long as three and four months if the indication is strong enough. On numerous occasions we have been able to completely restore and in many others partially restore the secretion after six or eight weeks. After three or four weeks but little difficulty is experienced as a rule if persistently attempted.

For practical purposes the increase of breast secretion or restitution of breast feeding depends upon creating a demand upon the breast for greater production. This may be done by one or both of two methods. The simplest way is through the nursing of the infant. The effect produced by the sucking of a vigorous child cannot be equalled by any other procedure. However, it is not often that we can get the stimulation from this source for in the cases where we wish stimulation we do not have a strong vigorous infant. If another child is available nothing better can be done than to place him at the breasts for a time. Here again we may have trouble for the infant will not nurse long while obtaining no satisfaction.

Zlocisti has suggested that we put the husband to the breast in these cases. While this undoubtedly would be a very effective method it is just a trifle too unesthetic to prove extensively practical.

Manual expression of the breasts has given the best results in our experience. In following this out one must observe a few details of technique which are very important. If we recall the anatomy of the breast we remember that from each lobe proceeds an excretory duct. These ducts converge into ampullæ which act as temporary reservoirs. In our expression we must seek to empty these reservoirs.

This is best done by placing the thumb and forefinger, one on either side of the nipple, just outside or a short distance behind the border of the areola, squeezing downwards and then pressing forwards. It is not necessary to use force or cause pain. Even extensively fissured nipples may be expressed with comfort. This procedure is repeated in a manner exactly similar to the milking of a cow. It is of no avail to go far back on the breast, in fact this interferes with the success of expression. If this is properly done, milk can be obtained from the breast immediately after an infant has nursed. There is no comparison between the effectiveness of manual expression and such devices as breast pumps. An amply secreting breast will often dry up when being emptied by breast pumps. Manual expression makes it possible to obtain considerable quantities of milk after the most complete evacuation by a pump. Breast pumps may occasionally be of value in removing milk from overfilled breasts but are of no value and are often extremely detrimental in the increase of breast supply. Electricity, massage, hyperemia, steaming, etc., are of no value in the stimulation of breast secretion.

Regularity of expression is important. The best plan for the increase of breast supply is to have the infant nurse both breasts at the regular nursing periods and then to practice complete manual evacuation afterwards. It is usually but a few days before a definite and continued increase in the breast supply becomes evident. Such rapid results, however, are not to be expected where the secretion has been stopped for some time. In these cases we do not expect much result before a week, and frequently 10 days or two weeks are necessary. The progress is usually gradual, starting with a few c.c. and working up. One must be cautioned against impatience and the mother particularly must be frequently reassured during the first few days of the attempt. Not infrequently restitution will be so complete as to sufficiently nourish the infant, even after total weaning for two or more months.

The seeming hardships connected with the carrying out of these methods is not at all out of proportion to the benefit derived by the infant for the even small amounts of breast milk sometimes obtained.

Of considerably more importance than the restitution of the supply in a dried up breast is the increase of milk by a breast which is producing an insufficient amount. It is only occasionally that we desire to restore breast milk to a mother but it is a matter of daily occurrence to desire a more ample secretion on her part. Ordinarily a breast-fed infant is nursed at one breast every three or four hours. If then a child that is being nursed in this way does not gain properly in the absence of gastro-intestinal symptoms we are reasonably sure that he is not getting sufficient food. The first step is to have the baby nurse both breasts instead of one at each feeding. In a very large number of cases this will in itself take care of the deficiency. If it does not, the next procedure is to practice complete manual expression of the breasts after the infant is through nursing. The technique and physiology of this method have been explained above. We must simply remember that the more milk we remove from the breasts the more there will be present. The expression should be diligently carried out after each nursing so that every possible drop of milk is removed, and the maximum demand made upon the breast. There is no case in which some increase in the breast supply will not take place and in by far the greatest majority of cases, the increase will be sufficient to nourish the infant. In case it is not, a determination that can only be made after 10 days' to two weeks' trial, complemental feeding must be resorted to, as described in another section.

WEANING

The indications for weaning have been taken up in a preceding chapter, but it should be repeated here that active tuberculosis in the mother is the only positive indication before the sixth month. All others are purely relative and usually possible to overcome. Pregnancy, menstruation, "colic," "failure of the milk to agree" with the child, are under no circumstances to be regarded as cause for weaning. In most cases breast feeding should be carried out for 9 or 10 months unless this period falls in the warm summer months. In that case, the nursing should be continued until fall. The technique of weaning is simple. It is much the best plan to begin to add food to the infant's diet in addition to the breast, two or three months before total weaning. The amounts and number of these foods are increased and the nursings gradually dropped until finally no more are given. There is wide variation of opinion concerning the character of the foods to be substituted. Some think that nothing should be given during the first year except milk. Others are feeding cereals, vegetables, etc., as early as the fourth and fifth month. Doubtless an intermediary method, such as is employed largely throughout this country now, is better. The addition of well-cooked cereal before one or two nursings at the sixth or seventh month, followed a few weeks later by carefully prepared vegetables and broths, has proved very beneficial in our practice. When weaning is desired one nursing should be dropped every week or 10 days until all are replaced. We feel that the addition of these foods before weaning is complete assures the safety of the child's diet later and at the same time guards against rickets and the demineralization associated with overfeeding on milk. A lengthy discussion of this subject, however, is out of place here. The point we wish to emphasize is that weaning should be gradual and complete by the eleventh or twelfth month.

There should be no digestive disturbances due to weaning. Disturbances at this time are due to the indiscreet addition of other food, or to infectious processes, not to the withdrawal of breast milk. At the ninth month most normal babies will tolerate whole cow's milk. Some children present food idiosyncrasies in early life and for this reason new foods are added slowly and only one at a time so as to be able to locate the cause of any disturbances. Eggs and fatty foods are to be avoided until later, particularly in children with the exudative diathesis.

In case some digestive trouble does develop during weaning it is usually advisable to revert to the breast feeding entirely for a time until the trouble is past. This is especially true of the diarrheas occurring in the summer time, and explains why total weaning is inadvisable at that season.

The breasts need practically no care during weaning if the system of gradually dropping the nursing is used. In this way the breast supply is able to adjust itself to the needs of the child and by the time nursing is wholly stopped the secretion has been so reduced that no trouble whatever is experienced. A little extra care in the matter of cleanliness if the breasts should become congested may be wise, but in the great majority of cases is unnecessary.

HYGIENE OF THE NURSING MOTHER

So many misconceptions of what the nursing mother should or should not eat have been handed down for generations that many of them are still rife and participated in by many medical men. Many young mothers have made nursing a great burden and have interfered with their health and their ability to nurse by attempting to live on unpalatable gruels and other liquids. As far as can be determined by extensive experiments, particularly on cattle, the diet has very little to do with the quality of the milk. Eckles has shown that a cow on a diet insufficient to nourish herself will continue to secrete milk and even produce more butter fat than before, at the expense of her own body. It is now the general policy to allow great freedom in the mother's diet. "Anything that is good for her husband" is a frequent and excellent way of stating the situation. If she likes pickles and sauer kraut there is no reason why she should not eat them in proper quantity. The only requirement should be that she take sufficient food to supply her own body needs as well as the infant's and drink about a pint of extra fluid daily. The form in which the food is taken is relatively unimportant for it is all broken down during digestion to similar compounds and the milk formed from the constituents of the circulatory blood. The freedom of a general diet removes one of the burdens of lactation and is effective in aiding lactation and in producing an abundant supply of milk.

The habits of the mother require a little more care. It is important that she should be regular in her eating and sleeping. A little more rest should be obtained than at other times and excessive emotion should be avoided as much as possible. If some relief can be obtained from household cares it may help but many of the best nurses are the ones who are busiest in their own homes. A regular life without worry is the ideal condition.

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CHAPTER XXIII

ARTIFICIAL FEEDING OF INFANTS

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HISTORY

Artificial feeding, in the sense of feeding a young infant exclusively on food other than breast milk, is a matter of very recent times. Only in the last four or five decades can one even speak of a true science of artificial infant feeding. Among the ancients artificial feeding in this sense was practically unknown if we may judge by the records handed down to us. In vain does one look for a single reference to the subject in Hippocrates, or Galen, or the Arabians, or any of the ancient physicians or philosophers.[1] Plato recognizes breast feeding, either from the mother or from a wet nurse, as the only form of feeding. Soranos of Ephesus (in the second century, A.D.) once mentions artificial feeding only to find fault with "foolish people who begin too early" and advises putting it off at least to the sixth month.

The reason for a lack of interest in anything but breast feeding among the ancients is evident enough. Artificial feeding has always been recognized as a substitute for breast feeding only when it becomes necessary to resort to it because of a dearth of mother's milk. Among primitive peoples there is no such dearth because nearly all mothers can nurse their babies. Among the ancients, social conditions were such that slaves could easily be procured to nurse babies whose mothers were unable, or disinclined, to nurse them. The fear of syphilis, too, did not yet exist. In Rome it was customary for Patricians to buy slaves to serve as wet nurses even before the baby was born and we know that in the time of Augustus the Plebians hired wet nurses who stayed at a wet nurses' bureau in the Forum Olitorium.[2]

That mixed feeding, and possibly to some extent artificial feeding alone, was, however, not altogether unknown to the ancients is evident from the finding of utensils that could have been used only for nursing, and also from meager references in the literature to these vessels and also to the kind of food used. A portrait in relief, now in the Louvre, of a mother and child, the mother holding in one hand a very modern looking bottle and in the other a stick to mix the feeding, has come down from the ruins of the palace of King Sardanapolis of Nineveh, who died in 888 B.C.[2] In ancient coffins of Greek babies have been found vessels closely resembling some still in use as nursing bottles in modern Greece. Recently unearthed infant coffins of

Roman times have likewise revealed great numbers of bottles of glass, or of earthenware, not unlike those of Greece. Some of these resemble not a little our present bottles, but commonly they are shaped more like a jug; or a pitcher with a spout; or a tea-pot; or like the modern drinking cup with a spout used by bed patients. Soranos, too, gives evidence as to the existence of such bottles when he says in discussing weaning: "If the child is thirsty he must not be given milk between meals, but water, or a diluted wine from an artificial nipple." From all of this we know that in ancient times babies were fed artificially but in all probability only in conjunction with breast feeding or only in later infancy. That newborn babies were ever reared wholly artificially is extremely doubtful.

The evidence from the records concerning *the nature of the foods used* gives us no clearer knowledge on this point. From Ancient times down through the Middle Ages all references to food for babies include always the same few things that evidently were traditional. Among the Hebrews we find in the Old Testament (Isaiah, chapter 7, verse 14): "A virgin shall conceive and bear a son, and shall call his name Immanuel. Butter and honey shall he eat." From the Greeks and Romans we have references to "honey, milk and honey, pap with honey, and butter."

Among the Romans there is reason to believe that mothers of the later Empire to some extent departed from the ancient custom of nursing their own babies or at least procuring a wet nurse. We are told that good Antoninus Pius instituted rewards for successful nursing mothers as a stimulus to nursing, and as a check to artificial feeding. The practice of feeding babies from the bottle apparently became common enough in Rome that "assæ nutrices," or dry nurses, who specialized in the artificial feeding of babies came into existence, but little is known as to the ages of the babies so fed, or as to the results.

The darkness of the Middle Ages reveals nothing on the subject until it is suddenly illuminated by a faint light in the latter part of the fifteenth century. In 1473 Bartholomäus Metlinger,[3] a German physician in Augsburg, published the first edition of his Kinderbuch, probably the oldest Pediatrics in existence. The part dealing with artificial feeding is very brief but is of unusual interest as it gives us an idea of the status of the subject in that remote time. If the mother, or wet nurse, does not have enough milk for one reason or another, "then," Metlinger says, "the baby must be given pap (*muss*) in proportion as it is needed to supplement the milk. Further, it must be known that the children must be fed only milk and pap until they have grown the front teeth. Then one can give them somewhat stronger food—bread softened in milk or in a meat or pea broth. And when they have grown older one gives them lean, well-cooked meat, but only very little for from much and strong meat grow worms. *But the natural length of time to nurse babies is two years.* If, however, during this time they want something else to drink except their mother's milk and crave other food than pap then one can give them from time to time a drink of water, or water that has been brought to a boil and again allowed to cool, or at times a well diluted wine." If they must be weaned it shall be done gradually. "If they are still young, *as for example under one year of age*, and one must wean them, then one gives them from a bottle (tütlein) or a little jug a goat's milk boiled with one-fourth part of water. And, too, one can make for them an electuary (Latwerge) out of white bread and sugar, as Avicenna says, on which it may suck: One takes one part of good finely powdered bread and one-fourth part of sugar and makes of this a sugar teat (Strützel) in accordance with the apothecary's art." Thus the pacifier dates at least to the fifteenth century, and if Metlinger's reference to Avicenna may be so interpreted even to the Arabians about the tenth century A.D.!

Metlinger[3] was familiar with the fact that overfeeding caused digestive disturbances, and recommended feeding intervals of "over one or two hours, depending on the size and build of the child, till the milk has passed from the stomach to the liver." "Indigestion," he says, "comes from an over-abundance of milk which the child cannot digest and especially so if it is strong and the child has a moist and weak little stomach. Dysentery generally comes during teething from poor digestion, from strong milk, from cold or from excessive fluidity and bad humidity." For the latter he recommends in the non-breast-fed child "skimmed goat's milk, and also almond milk extracted with water." For constipation he advises a laxative for the mother, or wet nurse, "especially if she too is constipated," and gives the baby raw butter to eat and a suppository "from the apothecary," or one made of hard, salted lard. Thus while this little book, with its quaint pathology and etiology, and even quainter treatment, filled with the names of a polypharmacy long forgotten, reveals mainly the crude beginnings of our specialty, it shows also that many of the things since established by our modern science were then known empirically.

Relatively little progress was made during the next three or even more centuries. That artificial feeding was becoming more common is evident from a poem by Hans Sachs, the Nüremburg Poet (1544),[2] in which he says when a mother has a baby, she must have, besides a crib, also "milk, flour, and pans for the baby, a nurse maid and a little bottle." How discouraging the results were may be established from a report by Raulin.[2] In Rouen 132 foundlings were fed from 1763 to 1765 "on diluted milk, gruels and broths, with sugar water and apple juice as a regular drink after the third month." Of the 132 babies only five survived the two years! From various sources we are also told that at this time there were in different countries, but especially in France, dry nurses ("nourrices seches") who, as in Roman times, specialized in feeding babies before and after weaning, taking them into their own homes in the suburbs or adjacent villages. Many of these we are told were "Godless murderesses of children," reminding one of the later denunciations of "baby farms" by Zola. That some of them, however, were more successful and more deserving is witnessed by an epitaph in Bésancon which reads: "Here lies Mme. X who brought up successfully more than 60 infants on the bottle." That some ideas of modern hygieng were gradually evolving is evident from the construction of certain bottles that facilitated cleansing. Also in 1802 Sternberg writes that "the bottle must be kept scrupulously clean as well as the sponges and linen cloths (that were used as nipples) to prevent the formation of acids that cause vomiting, stomach-aches, sore mouths, green diarrhea, colic and wasting." He advises, however, that a "healthy person shall first suck at the nipple before giving it to the baby so as to be sure that it draws well," a procedure that is not wholly inexcusable when one considers the nature of the nipple.

A brief review of the *development of the method of giving the food* to the baby is also perhaps of interest in this connection. The method of direct nursing from the udder of the nursing animal, goat, cow, or ass, has been in vogue as a custom among peoples and also as a therapeutic measure from most ancient times down to the present time. This is discussed elsewhere. The evolution of the bottle has been studied exhaustively by Brüning[2] (1908) to whose interesting monograph on the subject I am indebted for some of the data of the earlier and for all of the present parts of this review of the history of artificial feeding. We have seen that among the ancients various utensils of various shapes and material were used. Utensils similar to those of the Romans as one might expect were doubtless used down to the Middle Ages. During this period, and probably even much earlier, the spoon was used to a large extent and both in German and French the expression "to bring up on the spoon" is still synonymous with artificial feeding. The finger, too, was used for the thicker food, a custom not yet extinct. In the thirteenth century we know from a number of references that the bottle in common use was made from an ox horn. One such reference is of peculiar interest as it also gives us an idea of the prevailing customs as to the feeding of babies and even older children. In the autobiography of Thomas and Felix Platter, one of them complains: "After my mother had borne me, her breasts became sore so that she could not nurse me, and I never had any other breast milk as my dead mother herself told me. I had, therefore, as is customary in this country, when babies are weaned, to drink cow's milk through a little horn; for one does not give the children anything to eat till they are often four or five years old, except only milk to drink." The small end of the horn had a little opening over which a double layer of parchment was tied like a finger cot. Such horns were current in England, France, and Germany for several centuries and are said to be used in Finland to this day.

In the *fifteenth century* we know from Metlinger and others that jugs, and cans or cups with spouts with or without handles, made of wood, later of earthenware, tins and other materials, were in common use. Such vessels were used down to the eighteenth and even nineteenth centuries, and are said to be in use to some extent to this day in Holland and Italy. In the *sixteenth and seventeenth centuries*, the period of the highest development of wood turning, feeding vessels of wood came into common use which more or less closely resembled bottles. In the early part of this period bottles of tin, and even of silver, were used to some extent but these seem to have been used most extensively in the eighteenth century in all of the leading countries of Europe. In the *eighteenth century* porcelain bottles were used to some extent only to be replaced in the latter part of that century by bottles made of glass, first mentioned by Raulin in 1769. These were of various shapes and designs and were often highly ornamented. In the latter part of the *nineteenth century* (1868) "Maw's feeding bottle" was introduced in England as a great advance in construction. This type of bottle, with a rubber nipple, guarded by a disc of ivory, bone, or other material, connecting with a rubber tube, which in turn connected with a glass tube held in the neck of the bottle by a perforated cork and reaching to the bottom of the bottle, is still occasionally encountered, though its chief usefulness now is "to point a moral and adorn a tale." The direct application of the nipple to the bottle is a matter of recent times; while in the last few centuries a sort of empirical hygiene ever more influenced the evolution of the bottle, it was not until after the advent of the bacteriological period that the ability thoroughly to wash and sterilize by boiling both the bottle and the nipple came to be the dominating influence in construction and shape.

In the earliest jug and can period the food was simply forced into the mouth through a spout. Later nipples were made of sponges, fastened into the bottle and covered with parchment, linen, leather, or chamois skin. From earliest time down even to 1840 we find mention of the use of cow's teats, specially prepared, as nipples. An early observer of the ox horn period complains of them because even if kept in cold water they soon decomposed! The glass bottles were early surmounted by a tin nipple that could be unscrewed. Cork nipples were also used, especially perforated and with a glass tube leading to the bottom of the bottle. In the latter part of the nineteenth century came the rubber nipple of Maw's feeding bottle, only to be replaced in recent times by the modern nipple attached directly to the bottle.

Not until the *latter part of the nineteenth century* do we find much relief from this long period of crude empiricism both as to the matter and manner of feeding. We enter then what Biedert has called the "chemical period in which the endeavor was made gradually to base the fundamental principles of nutrition upon chemical investigation of food stuffs, and which reached its acme in Liebig."[1] The work of Biedert himself, beginning in 1869, forms the transition to the recent period in which infant feeding has been placed on as firm a scientific footing as any other department of medicine. Biedert's chief contributions to the subject were: Extensive studies into the chemical and physiological differences between human and cow's milk; the elevation of the casein of cow's milk to the first rank as *the* disturber in artificial feeding due to a difference in the nature and especially the coagulability of the casein of cow's milk as compared with that of human milk; the theory of "a schädliche Nahrungsrest" (harmful food remnants), composed of undigested casein coagula that caused harmful digestive and bacterial changes; the theory that stool curds were undigested milk curds formed in the stomach; the first attempts to make a cream, milk, and sugar mixture (*Rahmgemenge*) of relatively the same composition as human milk with especial emphasis upon the reduction of the casein to about 1 per cent., with the idea that, so changed, it might approach human milk physiologically as well as chemically as an infant food; and finally, the establishment of the idea of a "Minimalnahrung" (minimal feeding), i.e., giving to an infant the minimum amount of food on which it can thrive normally. While many of Biedert's teachings have since been attacked and some disproven, he was nevertheless the dominating pioneer in modern scientific infant feeding which has accepted and elaborated many of his views.

In this country Thomas Morgan Rotch a little later followed an almost parallel course. His own investigations led him to the same conclusions concerning the digestibility of casein. Like Biedert he tried to modify cow's milk so that it would approach human milk chemically. For this he slightly modified and adopted the mixture that Meigs had used for some time. When this failed to act physiologically like human milk he varied the percentages of the different food elements in an attempt to adjust the mixture to the individual baby and thereby laid the foundations and later built the superstructure of Percentage Feeding that for many years dominated our own ideas of infant feeding (see "Percentage Feeding").

Another pioneer in this country was Abraham Jacobi whose influence extended beyond his own country. Along unconventional lines he exercised a powerful influence for nearly five decades. He never became a follower of the percentage school; always used simple milk dilutions instead of adopting what he called the "gospel of top-milk;" he assailed the intricacies of the current practice of artificial feeding by a characteristic epigram: "You cannot feed babies with mathematics, you must feed them with brains;" he advocated cane sugar, instead of milksugar, for the same reason that led others to abandon it later; he always boiled his milk; and his name will always be associated with the use of cereal water dilutions for their inhibiting action on the curd formation of cow's milk. Always an independent and original thinker and writer his views until more recent years did not find as much favor in his own country as they did in Germany, where his contribution on Infant Feeding in Gerhardt's epoch-making "Handbuch der Kinderheilkunde" earned for him the title of "der Amerikanische Kinderartzt."

In England the names of Barlow and of Cheadle stand out conspicuously in connection with the early development of our knowledge of certain nutritional disorders in infancy, notably rickets and scurvy.

In the last two decades the advances have been rapid and important. The work of Rubner and Heubner in establishing the nutritional needs of healthy thriving infants, based on metabolism experiments, and expressed in terms of calories per kg. of body-weight (energy quotient) and later corroborated by many observations on breast-fed babies, confirming and defining the earlier views of Biedert on minimal feeding, stands out prominently as a milestone in the progress of our knowledge of infant feeding. During this time has come especially the sharper evaluation of the different food elements in their relation to infant feeding and to the nutritional disorders with which the names of Czerny and Keller are inseparably linked. In most recent times the work of Finkelstein and his school as to the nature, causation, sequence, and consequent classification of nutritional disorders in infancy has taken the most prominent place in the forum of discussion. The details of the work of this period are interwoven with the text of the various chapters dealing with those subjects and do not belong in this brief review.

The advent of the bacteriologic period had, and still has, a profound but varying influence on the subject of infant feeding. In the early empirical period milk was doubtless first given raw, then boiled as we know from Metlinger and others of a later period. Advancing knowledge in bacteriology pointed to a bacterial cause of diarrheas, notably "summer complaint" and cholera infantum. Sterilization of all milk naturally followed. Milk is, however, profoundly changed in boiling or sterilizing; the natural food of the young animal is raw; and considerable evidence was produced that heating milk destroyed certain living properties and to some extent its antiscorbutic properties. A reaction against sterilization or boiling of milk followed, notably in this country. This led to certified raw milk on the one hand, and pasteurization on the other. In more recent times the problem has been complicated by evidence that raw milk and boiled milk are unlike physiologically as well as bacteriologically. This whole subject is now under active discussion and will be considered in detail elsewhere.

In the last decade the vitamines have come into prominence and have assumed an increasing rôle in all discussions concerning nutrition.

INTRODUCTION

At the beginning of a chapter on artificial feeding of infants it is well to bring into high relief the specificity of human milk for the human baby. So different is breast milk from any other food for the young infant that one may well think of the newborn baby as still vitally a part of its mother. Birth should change only the manner and substance, not the continuity, of maternal nourishment. The long period of helpless and immature infancy of the child, as compared with that of the young of many other mammals, removes it only a little above that of the marsupials such as the kangaroo and the opossum whose off-spring are born so undeveloped that they cannot live independently of the mother who transfers them at birth to the ventral pouch in which their nourishment is changed from the placental to the mammary type. The newborn infant needs its mother's milk, or that of another mother, to insure its normal development and even, except under favorable circumstances, its very life. Babies can and have many times been reared by all of us *without any mother's milk* and have shown no serious deviations from that normal development that regularly follows adequate breast feeding, and yet so exceptional are these cases that they loom big in our memories. The younger the baby the more necessary that at least a part of its food should be human milk. After the first few months the need becomes increasingly less urgent, but even then, though life in not endangered the baby, as a rule, will fail to present the peculiar bloom of health, and vigor, and activity that is inseparable from the breast-fed baby. Not that the artificial feeding of the young infant is so difficult that it is discouraging or depressing; on the contrary it is one of the most grateful chapters in all medicine. Nowhere is the response more prompt, more certain, and more appealing—nowhere is there a greater reward to the doctor for patient study and observation than in the domain of infant feeding. And yet the more fully one learns the fundamentals of the science, and the more nearly one masters the art of feeding babies artificially, the more one is impressed with the fact that to the young infant mother's milk is a true specific, and that during this early period, at least, all artificial feeding is a substitute that necessity alone imposes upon us.

In the development of our knowledge of so extensive and perhaps difficult a subject, in which the equation of personal experience and interpretation must necessarily form a large factor, there is always much difference of opinion, and ideas and theories are commonly urged with undue vehemence and often with intolerance. Rival schools spring up dominated by some master mind and often "confusion" becomes "worse confounded" for a time. Herod, himself, is commonly out-Heroded by his followers. From each new school, however, something of permanence in fact, or viewpoint, is left to take its place in the sum total of our knowledge. While the evolution of our knowledge of infant feeding has made steady and rapid growth in the last 40 or 50 years, revolution has at times hastened and at times retarded that progress. To the bystander the battle has always seemed fiercer, and less significant than to the participants. Indeed, the contentions of pediatricians about infant feeding were at one time a byword among non-pediatric medical men. Fortunate rather the subject that has enlisted such able and enthusiastic combatants and that has been worthy of the strife! To those within the field of activity the struggle has always been full of purpose and of meaning. And now as the smoke is clearing the object and the result become clear enough to all except those to whom scoffing is a habit, or an argument.

As in all other branches of medicine we must divide the science from the art of infant feeding. The science is general. It is made up of the accumulated and classified knowledge of all the able observers and workers in this field. It is the common heritage of all those occupied with the subject. It can be mastered by an intelligent graduate of medicine in a comparatively short time. At times it leads the way—again it is but the interpretation of what has long been known empirically. The art is individual. It is the application *de novo* of the science to the individual baby by the individual physician. It must be learned by experience, by patient and earnest and intelligent observation of the baby himself. It can be taught as the artist teaches his pupil, but the pupil must ultimately develop his own art. The art of infant feeding cannot be learned in the study, nor in the laboratory, nor yet in the unnatural environment of a hospital ward. It can be acquired in its fullness only in the home, or in the out-patient department, where the real baby exists in its normal environment and where it can be followed from month to month and even year to year. The test of an infant's food is not in what happens during that brief cross section of time during which it is under observation, usually for some acute illness, in a hospital ward, or in the consultation room, but in what the end result is, measured in terms of years rather than of months or days. He who has mastered only the science of infant feeding flounders before the first baby he tries to feed. He who has learned his art only from the laboratory, the consultation room, or the ward does not know the real baby in its normal habitat, neither its normal condition of health, not yet the incidence, severity, and nature of its natural morbidity. As well expect the naturalist to know the free animal who has only studied it in the laboratory or observed it in the cage.

We are confronted at the present by an anomalous condition in the domain of infant feeding. The general practitioner who really feeds the vast majority of babies, and who therefore has the greatest opportunity to develop the art, is probably more indifferent to the subject than he is to any other branch of medicine and that for obvious reasons. The attitude of the practitioner is commonly one of three. He considers the subject, first, of so little importance, or of such manifest uncertainty, that it can be left to the mother or to some thoughtful neighbor, or better still to a succession of trials of "baby foods" with their full directions for feeding a baby on the can itself, or in the accompanying literature, until one is reached that fits the assumed idiosyncrasy of the baby; or else he has a due respect for the subject, but considers it too difficult for him to master under his peculiar circumstances, and after floundering in an aimless course with what he has remembered from his student, or interne days, or with an unreasoned sequence of baby "foods" that will not fit, he finally sends the case to the "specialist;" or, thirdly, having while a student earnestly studied the fundamentals of the subject, he now applies himself diligently and intelligently to master it by observation of his cases, by extensive reading and by consultation with those who have gone before. With the last of these goes all of our sympathy—sympathy not alone for his proper attitude toward the subject—but sympathy also of that other kind, for the task that confronts him when he seeks for light in reading and in questioning those who speak with authority. I have recently gone over the chapters on artificial feeding of infants in most of the standard text books in several languages and this earnest practitioner was constantly in my mind. In the welter of ideas and of theories that confront him; of methods and viewpoints and depreciation of those of others; of flat contradiction, and changes of opinion concerning the things that would seem elementary and fundamental; in the riot of mathematics that makes the pages of some of them look like a treatise on higher mathematics, or astronomy; who can blame him if he finds with Hippocrates that "art is long and time is short and judgment difficult," and that the "baby food," at least, does not contradict itself nor question its own virtues.

The solution of this man's problem is not wholly evident at the present time. The present tendency of pediatric teaching offers only a failure to solve the problem. An army of workers in pediatrics is diligently unraveling the infant's metabolism, and ascertaining its minutest chemistry. The vast array of *clinical* babies that alone interest the practitioner and the vast majority of whom he feeds find themselves less interesting than their urine or their feces to many an investigator. The physiological chemist becomes not only the investigator but also the pediatrician, and the teacher of infant feeding, in spite of the fact that the real art of infant feeding must remain to him a *terra incognita* because he cannot enter it. The clinical pediatrician finds himself elbowed out of the arena and commonly feels his own comparative insignificance. Too, by the time he has mastered the art of infant feeding he has commonly become so busy with duties that seem imperative in the ancient struggle for existence of self and kin that he does not give adequate time to impart to the student the art that he has acquired. Relegated by custom to the larger formal clinic he is to a large extent withdrawn from that intimate contact of patient, student, and teacher, in which the art of feeding can alone be taught effectively. The future will find some satisfactory division of pediatric teaching between the physiological chemist and the pure investigator, on the one hand, and the clinician on the other, each frankly cognizant of his own place and of his limitations. Always in pediatric *teaching* at least one must remember the student who is to be the future practitioner, and also the army of babies to whom he alone will minister. If the clinician for the time finds himself in the cold the future will bring him back to take his place as the real teacher of the *art* of infant feeding, for in our zeal for infant feeding we must not forget to feed the baby.

In the rapid and enormous development of our knowledge of the subject within the short space of a few decades the natural tendency has been toward greater complexity rather than toward greater simplicity. In the natural evolution greater uniformity of opinion will prevail—never that complete uniformity that makes for stagnation—but that uniformity that results from the general recognition of that which is true and from the disproval and elimination of that which is not true—and this makes for simplicity. To him who can read between the lines the signs are not wanting that we have entered an era of greater simplicity and unity of opinion in infant feeding, "a consummation devoutly to be wished."

THE PROBLEM OF ARTIFICIAL INFANT FEEDING

Nature tells us in no uncertain language what food a young infant should have. Practically without exception young infants thrive luxuriantly on mother's milk. If the mother has no milk they will thrive equally well on an abundant supply of milk from some other woman. If all mothers could nurse their babies adequately, or if wet nurses were as plentiful and productive as cows, the problem of feeding the young infant artificially would cease to exist. While there is reason to believe that in the future an increasing rather than a decreasing number of mothers will be able to nurse their babies, the fact confronts us that the mother who has enough milk for her baby till the fifth or sixth month is in the great minority. With the relatively small number of dependent mothers and especially mothers of illegitimate children, in this country, wet nursing is not widely applicable and is commonly employed only in those desperate cases that have failed to do even fairly well on artificial food. The scarcity and high cost of wet nurses, and the inconvenience attending their employment have almost restricted their use to the well to do, and to hospitals where a number of very sick cases can be tided over the dangerous period by a single nurse. The ghost of syphilis, too, now haunts the banquet, in spite of the Wassermann test.

The food of the young infant must be *liquid*. When the lack of mother's milk first forced the necessity of seeking some other food analogy naturally led the way to the use of the milk of some other mammal. All subsequent experience has only sharpened the view that milk must form the basis of all food at this period. Milk alone contains in suitable form the fundamental food elements necessary to sustain life and promote growth in early infancy. No other fat, or protein, or combination of salts can even approximately take the place of those of milk. The newer investigations of McCollum[4] and others into fundamental problems of nutrition bring home more than ever the fact that milk is an essential in infancy. The younger the infant the more imperative the demand. During the first few months it is almost impossible to maintain life without it. After the fourth or fifth month when other food can be given in greater abundance and variety, milk can be dispensed with if absolutely necessary but one would hardly know how to proceed and the baby would almost invariably suffer. In the latter part of the first year the problem, if forced upon us, becomes relatively easy but even then milk products can hardly be excluded without detriment to the baby.

The milk of a variety of mammals is serviceable in feeding babies. The choice is commonly determined by geographic and economic conditions. With us and in nearly all countries of Europe the cow is naturally the mammal chosen. In certain countries, like Cuba, Mexico, Turkey, and those of the Tropics, where the goat is the common milk giver, goat's milk is used for feeding babies. Ass's milk seems peculiarly adapted to young babies but obvious reasons prevent its extensive use. The milk of the llama, the camel, the reindeer, the mare, and the water buffalo can all be used for this purpose and some of them are. Unfortunately, the milk of each kind of mammal is specifically fitted to its own young, as Chapin[5] has so well elaborated, and commonly is quite unfitted in its natural state to that of another. Thus the calf that must rapidly be prepared to eat coarse solid food such as corn, oats, hay, and grass and that in large quantities to get the requisite amount of nourishment, receives from its mother a milk that forms huge, tough, leathery curds in the stomach and that is ultimately anything but a liquid food. The baby, on the other hand, in its relatively undeveloped state requires for a period of months both for its manner of taking food and also for its peculiar stomach a food that is liquid when taken and that remains practically a liquid in the stomach, and its mother's milk meets all of these requirements, forming in the stomach only a very fine, soft, flocculent coagulum. The fats and the proteins of the two milks differ essentially in infantile digestibility and the proportional distribution of the salts is vitally different.

It was soon discovered even by the earliest observers that simply feeding an infant on straight cow's milk was only exceptionally successful, and then only with older babies. The earliest attempts at adaptation consisted in diluting the milk with water because it was "too strong" for the baby, and later adding sugar, probably at first rather to sweeten it than to enhance its diminished food value. These first crude attempts were still found quite inadequate except in a few cases. All of our later efforts to adapt cow's milk to the baby have been refinements of these earlier efforts. *The essential problem of the artificial feeding of infants, then, is so to modify and to supplement cow's milk, which was intended by nature for the herbivorous calf, that the baby with its natural limitations will thrive on it, as nearly as possible, as well as it does on its mother's milk.*

COMPARISON OF COW'S MILK AND HUMAN MILK AS AN INFANT FOOD

The detailed consideration of the chemistry and biology of milk has been given in Chapter XXI, and only such facts as are necessary to an adequate discussion of the present subject will be presented here. Breast milk flows slowly, in response to just the effort that is normal to the baby, warm, living, amphoteric, and pathogenically germ free, directly into the baby's mouth at each feeding. The amount is regulated by a natural process of adjustment between the baby and its mother. If it deeds more it will empty the breast more thoroughly—if it needs less it will empty the breast less completely. The former is the greatest stimulator to milk secretion —the latter quickly lessens the amount to the proper level. If the amount of milk is adequate the mother and baby will soon meet in a happy medium ground and will continue there. Cow's milk, on the other hand, is drawn into vessels, and stands for hours, even days, before it is given to the baby, in quantities and by methods that are all determined by human calculations, not by Nature. It becomes distinctly acid in reaction, teems with bacteria, is either kept very cold alone for some time, or else is also heated to a point that destroys some of its living properties and changes its chemistry. It has, furthermore, a different chemical composition, a fat that is harder to digest, salts that occur in different proportional distribution, and a strinkingly different reaction to rennin. The part each of these may play in the comparative digestibility and tolerance for cow's milk as compared with human milk will be discussed later. Over and above these physical, chemical, bacteriologic and physiologic differences there seems to be some as yet imponderable biological difference that makes of human milk an absolutely specific food for the baby while cow's milk is specific only for the calf. Wherein this difference lies we are as yet wholly in the dark. Hamburger attempted to explain this difference by assuming a specific foreign protein reaction on the part of the baby to cow's milk protein taken *per orem* into the alimentary tract, analogous to the reaction that takes place when a foreign protein is injected subcutaneously, that is, directly into the body. He found that if cow's milk protein was injected directly the serum of the baby acquired the property of precipitating cow's milk protein, and not that of human milk. It was necessary to assume that the protein of cow's milk had an unfavorable effect upon the intestinal mucous membrane causing it to become pervious to the undigested *foreign protein* of the milk, and leading to the same reaction as if it were injected directly. There is little if any evidence that this ever takes place and Hamburger's theory of a harmful foreign protein (*artfremdes Eiweiss*) has now no longer any standing.

COMPOSITION OF COW'S MILK AND OF HUMAN MILK COMPARED

The food of all animals is made up of five food elements, fat, protein, carbohydrates, salts and water. The purpose and fate of each of these has been detailed in Vol. I, Chapter V. In addition to these food elements there are at least three other essential food constituents as yet not fully classified or identified, and all of these are well represented in milk. One of these is necessary to prevent the occurrence of scurvy. The other two, the "fat soluble *A*" and the "water soluble *B*" of McCollum, are essential to normal life and the former must be present or the young animal will not grow. It is not difficult to believe that these may have a yet wider significance than is now appreciated, especially in infancy, or that others of a similar nature may be discovered that are equally vital. For our present purpose it is necessary to consider only the five usually accepted elements.

The following table shows at a glance the comparative approximate composition of human and cow's milk:

	Human milk, per cent.	Cow's milk, per cent.
Fat	4.00	4.00
Sugar	7.00	4.50
Protein	1.50	3.50
Salts	0.20	0.75
Water	87.30	87.25
	100.00	100.00

With our knowledge of the chemistry of human and of cow's milk quite developed as it is it would seem the natural thing to do, as was hitherto considered adequate in studying problems of nutrition, to so modify cow's milk that it approaches quite nearly in chemical composition alone to mother's milk. This was the aim of the earlier scientific students of the problem such as Biedert and Rotch. Within certain limits such an approximation can be made by mixing cream, milk, skimmed milk, milk sugar, water, and certain salts. Unfortunately the composition is not adequately the same, or else, as has been suggested, there is more probably some other yet unknown specific difference, for the baby, the final test of all feeding, does not interpret this synthetic food as physiologically identical with its natural food. The well-known fact that if only a small amount of human milk is added to the food of an artificially fed baby the baby will tend to thrive as if fed wholly on the breast would seem to confirm this view. Efforts to make a "one food" like human milk for all babies out of cow's milk along chemical lines, not unlike the Alchemist's dream of old of making gold from baser metals, have largely been abandoned except for sporadic attempts that will be considered later. As the Alchemists, however, led the way to modern chemistry so these attempts have led to sharper definitions of the chemical and physiological problem of feeding cow's milk, and further investigation may yet lead to the desired *El Dorado.*

Practically, instead of trying to effect a chemical approximation, we find resort to a large number of quite unnatural, but physiologically effective expedients, such as the use of barley water, lime water, starch and dextri-maltose preparations, buttermilk, *Eiweiss milch.*

and a host of others that would seem quite foreign to any rational methods of feeding. *In other words, our further aim in adapting cow's milk to the baby is not to make it like mother's milk chemically but to make it, by modification and supplementation, act as nearly as possible as adequately as an infant food as does its mother's milk.* While our food mixtures, however, often bear little resemblance physically and chemically to human milk, we nevertheless always keep before us the composition and properties of human milk as our standard, and depart from these only so far as the already unnatural food with which we are dealing makes it incumbent to do so.

The **fat** of the two milks is nearly the same in amount. There is, however, considerable difference in the chemical composition. While in both the greater part of the fat is in the form of neutral fats, human milk contains a greater amount of oleic acid than does cow's milk. The greatest difference lies in the fact that cow's milk contains about 7 times as much of the volatile fatty acids as does human milk (10 per cent. of the total fat as compared with 1.50 per cent.). The fat globules are probably on an average somewhat larger in cow's milk than in human milk.

The **carbohydrate** is in the form of lactose, or milksugar, in both milks and there is no known difference between them. Human milk contains roughly one and one-half as much lactose as cow's milk.

The **proteins** of the two milks offer the most striking quantitative and qualitative differences. Not alone is there more than twice as much protein in cow's milk as in human milk, there is also a proportional difference in the amount of casein, as compared with the amount of the soluble whey proteins, lactalbumin, lactoglobulin, etc. In human milk the soluble or whey proteins are about equal to or even in excess of the casein or coagulable protein, while in cow's milk the casein is from three to five times as abundant as the whey proteins. These figures vary to a considerable extent in reports from different sources because of the difficulty in accurately separating chemically the two types of protein in both human and cow's milk, but especially in the former because of the well-known difficulty in precipitating the casein of human milk. Whether the casein of cow's milk is identical chemically with that of human milk is not yet known. The same is true of the soluble proteins. That they are both identical biologically is evident from the fact that all the proteins of each milk give *specific* precipitin reactions with the blood of sensitized animals.

The **salts** likewise show marked differences. The total ash of cow's milk is more than three times that of human milk. In general, cow's milk contains more of each of the salts, but a relatively larger amount of calcium, because of the larger amount of casein, and of phosphorus, and a relatively smaller amount of potassium and iron salts than does human milk (for details see Chapter XXI). While cow's milk contains more phosphorus than does human milk only one-fourth of it, however, is in organic combination as compared with three-fourths in human milk. This is probably of considerable significance in those clinical conditions in which phosphorus plays an important role. The phosphorus in organic combination is probably in the form of lecithin and of nucleon, which are said to be represented in larger amounts in human than in cow's milk. Chemical differences in determining the amount of lecithin and nucleon in the presence of other phosphorus-containing bodies do not as yet permit of accurate conclusions on these points.

PHYSIOLOGICAL BEHAVIOR OF THE DIFFERENT FOOD ELEMENTS OF COW'S MILK AS COMPARED WITH THOSE OF HUMAN MILK; THEIR MODIFICATION AND ADAPTATION IN THE ARTIFICIAL FEEDING OF INFANTS

The casein, the fat, and the sugar and salts of the whey have each "strutted their day" as the "enfant terrible" of artificial feeding. No one of these elements can any longer be assigned the leading role. All of them play a part, sometimes singly, sometimes in combination with others, often all of them at the same time—now in one part of the stage, now in another. Some of the food elements cause trouble in that they are difficult to digest. This is notably so of the casein and the fat. Others cause in addition to this disturbances in metabolism. This is especially true of the fat and probably in a less degree of the salts. Again the proteins, the fats, and the sugar are all acted upon by bacteria either without or, what is infinitely more important, within the digestive tract. As a result we have putrefactive and fermentative changes either together or, depending respectively upon whether the protein, or the carbohydrate and the fat are chiefly acted upon, one predominating over the other.

Each food element when acting alone as a disturber in infant feeding leads to a fairly constant clinical picture. It would seem, *a priori*, a very simple matter to treat the different food elements as distinct entities, observing their effect on the baby, and altering them according to the indications thus given. While we try at all times to observe the effect of each individual element and to modify it in accordance with our observation, we can only succeed within certain limits. As a matter of fact it is commonly quite impossible to proceed in this manner and reach the desired result. The reasons for this are not hard to see. In the first place the symptoms arising from indigestion on the one hand and bacterial activity on the other are commonly too much alike for practical distinction. It is only too often quite impossible to tell whether a baby has a digestive disturbance alone; a digestive disturbance plus a gastro-enteritis, or an intestinal food fermentation; or an intestinal fermentation; or a gastro-enteritis alone. In the second place, it is by no means always possible to tell what food element is *primarily* responsible for a disturbance, or, what is often more important, what element or combination of elements is now mainly responsible for keeping it up. Thirdly, there is an inter-relation and inter-reaction between the different food elements that is of far greater importance than the very scant attention paid to it in the literature would seem to indicate. Thus, a baby can stand a relatively large amount of sugar, or of fat, if the other is kept low in anount, and especially if the whey has been removed. A baby that can stand almost no fat in an ordinary milk mixture will stand an enormous amount of it if the whey and sugar are largely removed as in *Eiweiss milch* (see later.) Again a baby will tolerate a much larger amount of sugar in condensed, or dried, or acidified milk, or in *Eiweiss milch,* than it will in an ordinary raw milk mixture. In the former group the casein is in a form that can produce no digestive disorder, in the latter it can do so, and will then drag the other food elements with it in a combined disturbance. In milk mixtures in the practical feeding of babies one can give much sugar if one gives relatively little milk, or vice versa. To give both in large amounts invites disaster, though the total food value may be less than in either of the other two cases. Fourthly, including in a way all of the others, is the common observation that if a baby has a severe digestive or fermentative disturbance its ability to digest all food is lowered, or for the time abolished no matter what food element was responsible for the disorder, or what combination kept it up. It is useless to lessen, or even eliminate, one or the other of the food elements—all must be eliminated for a short time and only restored again gradually to the extent that the immediately present condition of the baby's digestive tract will permit.

The problem then is not a simple one of regulating the amount and modification of each food element according to the indications presented by the infant. If it were so simple, or so definite, one could quickly master the whole subject. In the practical feeding of babies science is short and art is long. As some one has said, "one does not know how to feed a baby till one follows the hunch rather than one's scientific knowledge." The more one knows about the practical feeding of babies, the less precise and outspoken is one's knowledge of the relation of the different food elements to a given disorder in a given case—but with the greater certainty and speed can one reach the desired therapeutic end. That end, too, can be reached by a variety of ways that may be based on wholly different interpretations, but that all lead to the same favorable result. This alone explains the equal success of men of widely different views and viewpoints whose methods and mixtures have little resemblance to one another.

In spite of what has just been said it is necessary to know very definitely the *behavior in the infantile economy of the different food elements of cow's milk, and the effect of modifying them so as to influence that behavior*, for of such material only can the art of feeding babies be built.

The Fat.—The fat is represented in human and in cow's milk to nearly the same amount. Qualitatively, too, the differences hardly seem of such a nature that they would account for the great difference in digestibility of the two fats. Human milk fat causes very little trouble—cow's milk fat is probably the most difficult food element to digest and assimilate. It does not seem probable that the greater oleic acid content of human milk, not the greater richness in volatile fatty acids of cow's milk, nor yet the slight difference in the size of the fat globules, could account for this striking difference. Certain evidence that we have points rather to the fact that has received too little attention, that it is not the fats, *per se*, but the medium in which they are given that is perhaps the determining factor. Take a large part of the whey out of cow's milk and coagulate and finely divide the casein, and we have essentially a food (*Eiweiss milch*) that almost without exception the most delicate baby can tolerate—yet the amount and strength of the fat is nearly that of undiluted milk, an amount that represents many times what could be tolerated in a simple fresh cow's milk mixture. In other words not the fat alone, but the fat in the *milieu* of cow's milk, is hard to digest. The very interesting "whey-exchange" (Molken-Austausch") experiment of L. F. Meyer[6] confirms the same view. Meyer coagulated cow's milk and human milk. He then mixed the curd of human milk, which of course contained nearly all the fat, with the whey of cow's milk; and the curd of cow's milk, which contained the fat, with the whey of human milk. These two mixtures he fed to babies just convalescing from severe digestive disturbances. Those to whom human milk whey and cow's milk casein and fat were fed all gained and their digestive disturbances cleared up. Those to whom cow's milk whey and human milk curd were given failed to gain and some of them relapsed to their former condition. Identical results were obtained by others (Grosser, Erich Müller, Moro).[6] We know too that a larger amount of fat can be taken in boiled milk than in raw milk, probably because of the more attenuated and less harmful casein curd. We have here again illustrations of the inter-relation of the different food elements in determining the acceptability, or non-acceptability, of a given food element.

It is probably because of this inter-relation that cow's milk fat causes two wholly opposed disturbances, diarrhea and constipation. Thus on the one hand we know that fat indigestion can manifest itself as a diarrhea, with frequent acid stools containing fat curds, even though the condition may arise only secondarily to a carbohydrate fermentation. On the other hand, we have the fat, when given in excess and not accompanied by an acute disturbance, leading to a fat constipation with hard, dry, putty-like stools composed largely of the salts of fatty acids. Cow's milk fat is apparently in itself constipating, and remains so in a mixture such as dried or boiled milk, especially if the carbohydrate is kept low—a mixture that does not easily lead to diarrhea. If given in raw milk, or with milksugar, it too becomes involved in the diarrhea that is much more apt to occur.

There is perhaps no more constant symptom of fat intolerance of both human and cow's milk than is *vomiting*. From the composition of the two fats it is again not wholly clear why the fat of human milk causes so little vomiting, while that of cow's milk so much. The essential difference in this respect is evidently not so much one of relative digestibility, for fat is not digested in the stomach—but rather one of the length of time it takes the fat to leave the stomach. After a feeding of human milk with its 4 per cent. fat the stomach is probably emptied in two, or at the most three, hours. After a feeding of cow's milk with the same strength of fat the emptying is probably delayed to twice that time. The richer the food in fat the longer the emptying time and the more easily does vomiting occur. In a series of experiments that I conducted several years ago in which the behavior of a large number of modifications of cow's milk in the stomach was studied on an adult who could at will empty the stomach by vomiting, this fact was well illustrated.[7, 9] After taking a pint of cream (16 per cent.) very nearly a pint was returned from the stomach after two hours. The returned stomach contents "looked like a soft, rich yellow, greasy cottage cheese, with a very strong odor of fatty acids. . . In no other experiment was there even approximately so large a mass of curd and milk that had apparently been influenced so little." In a second experiment there was still present after five hours 4½ oz. of a "slimy, thickened, coarsely granular, creamy liquid, like a thin cottage cheese," that was "very offensive, with the characteristic rancid odor of fatty acids, quite identical with that of the 'sour stomach' of the vomiting baby." After seeing and smelling such contents returned from the stomach it is not hard to believe that babies vomit high fat cow's milk mixtures both because the stomach is never completely emptied otherwise and also because of the keenly offensive and irritant nature of the stomach contents. Another factor probably enters in. Milk rich in fat, and especially cream, if taken in relatively larger amounts becomes nauseating in itself. Thus the subject of these experiments could only with difficulty swallow and retain the pint of cream at the first trial—and so difficult was this with each succeeding time that only three successful experiments were made.

In adapting the fat of cow's milk to the baby little can be done that is of value except to vary the amount. It was once thought that gravity cream was more easily digested than centrifuged cream but this wants demonstration. The fat can be lessened by skimming the milk, or by giving less milk in a simple diluted milk mixture, or by using less cream in a cream mixture. It can be increased by using cream, or top milk, or more milk in the mixture. As has been indicated, the most effective method of adapting the fat to the infant is to modify the medium in which it is given. The essential point here is to give it in a medium that does not easily lead to indigestion or fermentation, i.e., by keeping the sugar low; by reducing the curd to a minimum by the various methods that will be detailed directly; and in some cases by reducing also the whey, that is, the salts as well as the sugar (Eiweiss milch and whey adapted milk). The common therapy consists in keeping the fat low and making up the deficiency with carbohydrates, as in the use of buttermilk, skimmed milk, and especially malt soup (Keller's "Malt Soup").

Homogenization.—An attempt has been made to make the fat more digestible by lessening the size of the fat globules which are said to be slightly larger on the whole than those of human milk. This process of *homogenization* consists in forcing the milk by means of a pump through a finely ground agate valve against a pressure of 3000 to 5000 lb. to the square inch. The resulting fat globules are so altered that they will no longer separate as cream, and are much smaller than they were. Homogenized milk, first used in France, has recently again been advocated, especially by Ladd[8] who reported very favorable results in a series of 37 cases of "malnutrition and indigestion" that seemed unable to tolerate cow's milk fat. In this series a further expedient to circumvent the difficult digestion of milk fat was resorted to, i.e. *the substitution of an oil for the milk fat.* Ladd added from 1.5 per cent. to 3.5 per cent. of olive oil to skimmed milk mixtures, then passing them through the homogenizer. Olive oil was especially chosen because it is made up largely of olein and palmatin as are the neutral fats of both human and cow's milk; and even more because it is practically free from volatile fatty acids that are represented in cow's milk fat to the extent of 27 per cent., and are held by some to account for the greater difficulty of digestion of cow's milk fat, as compared with human milk fat with only 2.5 per cent. of volatile fatty acids. The value of homogenization or of the substitution of homogenized olive oil has not been confirmed and remains to be demonstrated by further investigation.

The Carbohydrate.—*Sugar.*—The sole carbohydrate of all milk is milksugar, or lactose, and it is apparently identical with the milksugar of commerce. Milksugar cannot be modified in any way in infant feeding. One can only vary the amount—lessening it by reducing the amount of milk, or taking out part or all of the whey which contains the sugar—increasing it by adding commercial milksugar. Inasmuch as cow's milk contains less sugar than human milk and is furthermore usually materially diluted in early infancy, sugar is nearly always added to cow's milk mixtures to make up at least a part of this deficiency. Empirically it was learned rather early that milksugar cannot be added with impunity up to an amount represented by the 7 per cent. of milksugar in human milk, and various other sugars and also starch have been used as substitutes for the natural sugar in the hope that by using one, or several of them at the same time, a larger amount of carbohydrate could be given without leading to a clinical intolerance. Substitution, then, and not modification, is the only method of adapting the carbohydrate to the infant.

Milksugar.—Human milk is peculiarly rich in sugar. A quart contains 2¼ oz. of milk sugar or about 23 level teaspoonfuls. We cannot escape the conclusion that babies were intended to have a large amount of sugar. The natural corollary would seem to follow that the smaller amount of sugar in cow's milk should be amply supplemented by additional milksugar. It is one of the most certain facts in infant feeding, however, that the practical application of this principle leads to disaster. Milksugar can be given alone in large quantities without leading to serious trouble. In the medium of human milk we know that it occurs with remarkable constancy in large amounts and probably never causes any digestive disorder. When added to fresh cow's milk as an infant food it must be used with the greatest caution long before such an amount is used as is represented in human milk. It has in fact been assigned the leading role in initiating all digestive disorders that are fermentative in nature. Sugar intolerance is characterized by frequent, acid, frothy, irritating liquid stools. It must be borne in mind that in the digestion of human milk with its low protein, and very high fat and sugar content, fermentation predominates and the stools are normally soft, mushy, acid and non-offensive; that in the digestion of cow's milk with its very high protein and relatively low sugar and fat content, putrefactive changes predominate and the normal stool is formed, or pasty, alkaline in reaction, and has an odor of putrefaction. By keeping the protein low in feeding cow's milk the normal fermentative condition that goes with the digestion of human milk can be approached. When it is reached, however, the condition becomes pathological, for frequent, acid stools are always abnormal when one feeds cow's milk and are incompatible with favorable progress. It follows that in cow's milk mixtures used in feeding babies there is an inter-relation between the amount of milk and the amount of sugar. If one uses much milk one must use little sugar, and vice versa. One clinician who uses low protein and low fat mixtures cannot demand of another who uses concentrated simple milk mixtures that the latter should use as large an amount of sugar as he himself finds safe. The dosage of sugar cannot therefore be considered as an isolated factor, but must be considered in connection with the nature of the medium in which it is given, and with the degree of representation of the other food elements. The physical state of the casein, whether coarse as in raw milk, or finely divided as in dried, condensed, alkalinized, boiled, acidified, or precoagulated milk, is also a factor of great significance in determining the tolerance for sugar. As in the case of the fats we have here again an illustration of the fact that the tolerance of a food element depends upon the medium in which it is given, and upon an inter-relation with the amount and nature of the other food elements.

Other Sugars.—Because of the fact that milksugar when given to an infant outside of its mother's milk readily ferments to a dangerous degree and especially so in the presence of a gastro-intestinal disorder, it was a logical step both from empirical and from chemical reasons to ascertain whether other carbohydrates might not act less unfavorably. As a result of this step other carbohydrates are now used much more extensively in infant feeding than is milksugar. Nearly all of the Patented Baby Foods so popular in this country feature one carbohydrate or another, as superior to the natural sugar of milk. This is partly the result of Liebig's experimental work on malted flours, and partly an effort on the part of the manufacturers to meet the present demand for obvious reasons. Even among pediatricians, as so often follows, many have actually come to look upon milksugar as an unsafe food for babies under all circumstances. In one of the largest German clinics some years ago a young assistant told me that he had often been tempted to try milksugar sometime but that one heard such awful things about it that he had never dared to do it! And this in spite of the fact, as he told me, that his "chief" had at one time changed the sugar of 500 babies, after certain intervals, from milksugar, to cane sugar, and in turn to a dextrin-maltose preparation without being able to record any appreciable difference!

In the normal baby with a properly adjusted food and a normal digestion there is very little if any evidence that one form of sugar has any decided advantage over any other. Since milksugar is the normal sugar of milk, and so long as there is no definite evidence that other sugars are of greater value in the normal infant, it would seem the natural thing to do to use it in feeding babies. Since, however, it shows no tangible superiority over other sugars we can in no way object to the use of these other sugars, and especially so because they are "unnatural," for we have already departed far from the natural when we substitute cow's milk for human milk. In its natural medium milksugar is specific—in the unnatural medium of cow's milk some other sugar may well be more acceptable. Both cane sugar and dextrin-maltose preparations are said to be more fattening than milksugar and this offers an advantage in certain cases. How difficult it is to decide a clinical question of this kind, with so many factors entering into it, becomes evident when one scans the literature, or consults the practice of various clinicians of equal authority. Incidentally this very divergence of opinion and of practice can only confirm the view that the difference cannot be very great. In certain parts of this country milksugar is still used in the feeding of normal babies for much the same reasons that have been mentioned. In Germany and in a very large part of this country dextrin-maltose preparations have almost entirely replaced milksugar. These preparations, partly as a result of the literature on the subject and partly as a result of skillful and timely propaganda, have become so common with us that the up to date mother provides herself with certified milk and a can of a dextrin-maltose preparation and then calls in the doctor to tell her *how* to use it!

Cane Sugar.—There is no genuine evidence that *cane sugar*, which is much cheaper, should not be used as a routine as it has been done with great success in the Infant Welfare work of Chicago for a number of years. Jacobi, too, always advocated cane sugar in place of milksugar. The claim that cane sugar more readily leads to digestive, and especially fermentative, disturbances than does milksugar seems rather a matter of tradition than of clinical observation. All clinical evidence points rather to the opposite conclusion, if to any, as so long maintained by Jacobi. The objection that canesugar is too sweet rarely finds confirmation in the baby's taste. The statement that is still encountered that one must use only one-half as much cane sugar as milksugar because of the greater degree of sweetness dates back to a period when the importance of food values was less appreciated than now. The occasionally expressed preference for cane sugar as compared with beet sugar would seem to ignore the chemical identity of the two sugars. The chief indictment against cane sugar is that it ferments more readily than the other sugars. This stills lacks unexceptional demonstration in the normal baby, as does also the assumption that this tendency is harmful.

Dextrin-maltose Preparations.—Thus far we have considered only the relation of the various sugars to the normal baby. When the baby becomes abnormal, when it has a digestive disturbance and especially one of a fermentative nature, one's attitude toward the various sugars is materially changed. It seems well established that in these conditions milksugar, and also cane sugar, are less well tolerated than are the dextrin-maltose preparations such as Soxlet's Nährzucker, Mead's Dextri-maltose, Mellin's Food, and Horlick's Food. Because these carbohydrates are better tolerated in all cases with a tendency to diarrhea of fermentative origin, and can be given sooner and in larger amount during convalescence without danger of a return of the diarrhea, they have almost completely replaced the other sugars in the treatment of these conditions, and are always used in the special preparations such as *Eiweiss milch* that were devised to meet this indication. Dextrin-maltose preparations are also the carbohydrate of choice when the clinical indication is to give a relatively large amount of carbohydrate to replace a therapeutically diminished amount of fat, as for example in the treatment of fat constipation by the use of Keller's Malt Soup. Thus both in diarrheas, and also in milk constipation where one strives to obtain a more laxative action with a larger amount of carbohydrate than we could safely give of either milksugar or cane sugar, the dextrin-maltose is peculiarly indicated. Partly because of this practice, and also because the pathologic is commonly but little separated from the physiologic and easily succeeds it rapidly, these preparations have gradually replaced the other sugars in the feeding of the normal baby also, and no valid reason has ever been advanced why this should not be done. A further advantage in the use of dextrin-maltose preparations lies in the fact that it is commonly of benefit to use two or more carbohydrates in an infant's food for reasons that will be considered later. When we add dextrin-maltose to a food we have three carbohydrates represented, milksugar, dextrin and maltose, that have a different chemical and bacteriological behavior in the intestine and have different rates of absorbability.

Starch.—In the starch we have another carbohydrate that is wholly unrepresented in the baby's natural food and yet meets a most important and diversified need. It adds another carbohydrate that has a different action in several ways than have the sugars, and meets certain indications that are not yet clearly defined that are not met by the latter.

The question of how early we could give starch to a young infant was long a mooted question and there is still much divergence of opinion as to when to begin giving starch at all and how early it is advisable, or even safe, to give an appreciable amount. It can now be stated that in the newborn infant the amylolytic ferments of both salivary and pancreatic glands are present in sufficient amounts to digest as much starch as is represented in the cereal waters that are commonly the only form in which starch is given at this age. Clinical experience corroborates the presence and activity of these ferments. For many years I have used barley water almost exclusively as a milk diluent in earliest infancy and have never been aware of any unfavorable action. That the baby of four or five months can digest quite large amounts of starch such as farina, Imperial Granum, zwiebach, toast, etc., is a well-known therapeutic fact. The fact that Sauer and Helmholz have been able to feed successfully very young infants as high as 10 per cent. of farina in cases of pyloric stenosis, and that one sees infants fed in the first months of life on large amounts of Imperial Granum (75 per cent. starch) without evidence of starch indigestion would confirm the idea that even the youngest infant can digest as much starch as ever seems indicated for one reason or another.

Starch is used in infancy to meet several indications. In the first few months it is given commonly only in the form of a cereal water as a milk diluent primarily for the purpose of causing a finer curd formation in the stomach—only secondarily because of its food value. The action of cereal waters upon milk will be discussed more fully under casein modification. After the fourth or fifth month starch forms an important addition to the diet. The starch has in addition to its simple food value a clinically well known action that is not shared by the sugars. An infant that will no longer gain in weight on a simple milk, water and sugar mixture will commonly gain as soon as starch is added to the food, even though only in so small an amount as would be represented in a cereal water. This action seems not wholly due to the added food, but to some peculiar property inherent in the starch that causes a stimulation of metabolism of the sugars, fats, and salts. This probably explains the uniform improvement and rapid gain in weight at about the fifth month of the baby that has not done well on a simple milk, water, and sugar mixture if cereals are added to the diet.

The differences in the action of the different carbohydrates in the digestive tract and in the metabolism of the infant in normal and in abnormal conditions are probably in large part explained on chemical and subsequent bacteriological grounds. As the result of specific ferments the disaccharides, milksugar, cane sugar, and maltose are converted into monosaccharides that are not identical in the three sugars. Milksugar is changed into dextrose and galactose; cane sugar into dextrose and levulose; while maltose is split up into two molecules of dextrose. Dextrins are first converted into maltose and then into dextrose. The starches are first changed into dextrins and then into maltose and finally into dextrose. The disaccharides and the polysaccharides, like starch, are not absorbed as such while all the monosaccharides are. They seem, however, to differ in the readiness with which they are absorbed and utilized in the body and also differ in their fermentability in the intestinal tract. Dextrose is the least easily fermented and the most readily absorbed of the monosaccharides. It is moreover the normal constant sugar of the blood and can, therefore, be utilized at once without first being stored as glycogen in the liver. The levulose and galactose, which form half of the intestinal end products of cane sugar and milksugar respectively, are said to be more readily fermented and less readily absorbed, and must first be changed into glycogen and later into dextrose before they can be utilized in the body. This would seem to offer an explanation for the clinical experience that dextrin-maltose preparations are more acceptable to atrophic, or marantic babies, to weak babies with digestive disturbances, and to all cases in which there is a tendency to fermentation. Maltose for chemical and commercial reasons has not been available for infant feeding and is never used alone. It is always combined with dextrins which represent an intermediate stage in the conversion of starch into maltose. In fermentative disorders this is of considerable advantage, as we know clinically, probably because the simultaneous and more gradual conversion of dextrin into maltose, and maltose into dextrose, leaves less of the dextrose at any one time to undergo fermentation. It may also be of value for osmotic reasons. This probably offers a further explanation why starch is so often added with advantage instead of more sugar, because the digestion of starch requires a still further step, the conversion into dextrins. In very weak and very emaciated infants with feeble digestion this might, on the other hand, be a disadvantage because of the greater tax on the energy of the child.

The slower conversion of starch and dextrins probably accounts for the clinical experience that these carbohydrates are more constipating, or perhaps more accurately, less laxative than the disaccharides. At no one time or place is there so large an amount of fermentable monosaccharide in the intestinal tract as when the sugars alone are used. This would seem to account for the further clinical observation that in the use of dextrin-maltose preparations those in which the maltose is relatively high, such as Mellin's Food (maltose 59 per cent., dextrins 21 per cent.), are more laxative than those in which the maltose is lower and the dextrins higher as in Mead's Dextri-Maltose or Soxlets' Nahrzucker where the two carbohydrates are nearly equal in amount. The statement that starch is constipating is only true relatively. It is more constipating, or let us again say, less laxative, in the normal baby, or in the slightly disturbed older baby, than are the sugars. In the very young infant with a digestive disturbance starch may act more harmfully than the dextrin-maltose preparations especially if the greatest caution is not used. In no sense can one here use starch as a constipating agent. In the older infant with a similar digestive disturbance starch is commonly more easily tolerated than are any of the sugars.

The case of *barley water* deserves a special consideration in this connection. Barley water has become a sort of national institution in this country. When a baby has a diarrhea its milk is discontinued and barley water alone is given for a day, unfortunately only too often for many days. The diarrhea commonly ceases quickly and the barley water is given all the credit for the change. Naturally it has gained a reputation as an active constipator, not alone in the baby with a diarrhea but also in the normal baby. It does not concern us that water, or tea, so regularly used in some European countries will produce the same result in diarrheas *if only the milk is eliminated. Pari passu* with the idea of barley water as a constipator runs the idea that oat meal water is a laxative, although demonstration of a real difference is wholly wanting. That oat meal given as a cereal is more laxative than barley or wheat flour is probably true because of the greater coarseness and of the greater content of the non-digestible polysaccharide cellulose, but that oat meal water with these factors wholly eliminated is more laxative than barley water at least remains to be demonstrated. The reason for continuing the use of barley water in diarrheas is evident. It is at least harmless, it is a method of keeping up the child's water intake, it supplies a small amount of carbohydrate nourishment that may help to prevent an acidosis, and, more important than all, it satisfies the mother who would consider her baby starved if given only water or tea. Whether in very young infants it may not at times rather keep up than lessen a diarrhea is at least an open question.

In a very different manner barley water in common with all cereal or starch waters has a constipating effect by aiding in the digestion of milk. As we shall see later (see Casein Modification) starches by virtue of their colloidal nature when used as milk diluents cause the formation of very small and peculiarly porous casein curds in the infant's stomach. This action does not reside in any of the sugars nor in the dextrins.[7] As a result casein indigestion is eliminated, or reduced to a minimum, the casein is more easily, rapidly and completely digested and absorbed with a resulting tendency to constipation, or rather lack of indigestion and diarrhea. We find here again an illustration of the necessity of keeping in mind the interrelation of the different food elements in estimating the effect of any one of them.

The Protein.—*Soluble Proteins.*—The soluble or whey proteins occur both relatively and absolutely less abundantly in cow's milk than in human milk and especially so in the usual dilutions used in infant feeding. They present no known difficulties in digestion. They are therefore never purposely modified in infant feeding except as they are incidentally lowered in those foods in which the whey in which they are in solution is reduced in amount; or are relatively increased in those preparations in which whey is added to a cream mixture because it is desired to lower the amount of casein and yet keep the total amount of nitrogenous food undiminished ("Split-protein").

The Casein.—Cow's milk contains from four to five times as much casein as does human milk. Inasmuch as milk is coagulated by the action of rennin and of acids upon the casein it naturally follows that the two milks coagulate very differently. If cow's milk is coagulated in this manner *in vitro* it forms a homogenous semisolid jelly that will not fall out of the inverted beaker. Human milk under like conditions is only with difficulty coaguable at all and even then it remains a thin liquid somewhat thickened by the presence of a more or less discreet soft fine flocculent coagulum. Whether casein is a definite chemical compound alike in human and cow's milk is not known. That it is serologically specific for both species has been demonstrated. Whether the difference in coagulation is purely one of concentration of casein in the milk is consequently equally unknown. That it is largely a matter of concentration is obvious from the fact that the more cow's milk is diluted the more nearly does the coagulation approach that of human milk. That it ever completely equals it in like conditions of casein concentration is at least doubtful.

Recent investigations have shown how unreliable all deductions from such experimentation are as revealing what really goes on in the stomach.[7, 9] The stomach is not a passive organ. On the contrary it is in constant motion of a peculiar type. Other factors, too, such as the varying composition of the stomach contents in rennin, acids, salts, and, what seems of decided importance, mucus, doubtless have a determining influence on the curd formation. Hitherto our knowledge of what actually happens to milk in the stomach has been obtained only from animal experimentation; from the use of the stomach tube from the individual with a stomach fistula; and from observations on the vomiting baby. Animal experimentation has been notoriously unreliable as I have been able to demonstrate on the dog. Besides a dog's stomach is not a human stomach. Stomach tube results are worse than useless in studying the coagulation of cow's milk in the stomach because the curds that are formed are so large that only the smallest fraction of them can be returned through any possible tube.

The evidence furnished by the vomiting baby has been the most valuable but has been quite ignored and misinterpreted because it was always assumed that the vomiting baby had an abnormal stomach and that the resulting curd formation was abnormal. The baby that vomits mother's milk some time after feeding returns a liquid that contains at most fine, soft, flocculent curds such as we have seen in the beaker. The baby that vomits cow's milk, especially raw milk that is relatively little modified, will vomit either a turbid, acrid, offensive liquid, i.e., whey, or else in addition to this, as we have all seen many times, curds up to the size of a walnut and so hard that they will not break if thrown on the floor. This phenomenon is not to be interpreted as indicating an abnormal stomach, but simply as an accidental revelation of what we have every reason to believe is a perfectly normal reaction to cow's milk.

The vomiting of the baby gives us the clue to what seems the only possible method of studying the coagulation behavior of cow's milk in the human stomach. The stomach contents must be returned completely without injury to the curds and this can be done only by the act of vomiting. While the baby offers insurmountable obstacles to such an investigation, there are adults that can readily empty the stomach quite completely by simply passing the finger into the throat. With such a healthy young adult with demonstrated normal digestion I did some hundred experiments with all of those variations and modifications of cow's milk that are of interest to the pediatrician. That this individual gave a normal reaction is evident from the fact that in a few of them repeated on myself with the aid of apomorphine hypodermatically the result was identical; and even more so from the fact that the same experiments were in large part duplicated some years later by Bergeim, Evaard, Rehfuss and Hawk[10] who found in every instance that a like experiment gave a result identical with what I had obtained. For details concerning this work the reader is referred to the original papers.[9, 7]

Our present knowledge of the purely mechanical coagulation behavior of cow's milk in the human stomach as defined in these experiments, can be briefly summed up for our present purposes as follows: When a pint, or a quart (the result was always the same with the two amounts) of *raw whole milk* is taken into the stomach over a period of two or three minutes it begins to coagulate almost immediately if it is of body temperature. If the milk is cold coagulation is simply delayed till it is of a more favorable temperature for the action of the rennin. Within two or three minutes with warm milk fine, indistinct, soft, scraggly curds are formed. At the end of five minutes there is complete separation into nearly clear straw colored whey and more or less firm coagula of varying sizes. The curds are distinctly larger and harder after five minutes than after three minutes. At the end of 30 minutes the curds are both much larger and harder than after five minutes. In every experiment, with whatever combination or modification used, the curds become larger and tougher up to the end of the two-hour period after which they gradually become smaller, but, if anything, still harder. At the end of the two-hour period the curds were often so large that they could not be returned—although in one experiment after this interval a curd was returned that was "5 in. long and extended laterally at both ends, 2½ in. at one end and 1½ in. at the other, and was from ½ to ¾ in. thick throughout." At the end of five hours in one experiment following the taking of a quart of milk "there were still 10 curds present" each over 1 in. in length and from ½ to 1 in. in width. One curd was over 2 in. long.

All were rounded or almond shaped and firmer than in any preceding experiment. They closely resembled in every way except in color the *hard curds* of infant stools.

The increase in the size of the curds is due to the agglutination of smaller curds into larger masses resulting in bizarre shapes that are evidently the result of such union. If two recent curds that are formed *in vitro* are brought in contact they will at once coalesce without leaving a visible line of union. This tendency is gradually lost as the curds get older. In the stomach where the curds are constantly pressed together the result would probably be the formation of one large curd if it were not for the thick coating of mucus that is always in great evidence after an hour or so. As a resultant of these two factors, apparently, the curds always get larger by union with others for about two hours and then become smaller because such union no longer takes place and the curds are gradually decreased in size by peripheral digestion. For the first two hours the increase in size is greater than the diminution due to digestion.

As has been stated curds get firmer the older they are. This can also be observed *in vitro*. If milk is coagulated in a beaker it becomes uniformly solid. Gradually there is a separation of whey and curd, the latter contracting constantly in size and expressing the retained whey. After a time it becomes so hard that it closely resembles dry gelatine and can be broken up into hard, dry fragments.

Cow's milk is thus seen to be a food that is unique in two respects. It is ingested as a liquid food and becomes in the stomach a solid food. Secondly, it is not only a solid food but a uniquely solid food in that it becomes for some hours an increasingly solid food both as to the size and the density of the solid portions. Nearly all other foods that can be digested at all such as meat, eggs, potato, bread, etc. soften and disintegrate in the stomach and so permit of penetration by the digestive juices. The curd of cow's milk becomes larger for two hours, and instead of becoming softer and disintegrating in either the stomach or the intestine, it becomes increasingly harder. Curd digestion is, therefore, wholly peripheral, only the surface can be digested. In infants that are fed on raw milk the digestive process often does not have time to melt down this hard mass and it appears in the stool as the "hard curd." Cow's milk should, for these reasons, no longer be classed physiologically as a liquid food, but rather as one of the most peculiarly and uniquely solid foods that are ever used.

The amount of fat in the cow's milk has a decided effect on coagulation but even more so on the emptying time of the stomach. Fat free milk coagulates more quickly and completely than does whole milk, and forms a whiter, harder curd. The richer the milk in fat the slower and less sharply defined the separation into curds and whey, the yellower, smaller and softer the curd, but the harder to digest and the slower to leave the stomach. Cream remains for hours in the stomach with relatively little change in amount or in size of the curds, but with increasing acidity and offensiveness (see Fat for details).

The effect upon coagulation of cow's milk in the stomach of the time taken to ingest the milk was of peculiar interest.[9] Hoary with tradition and still with us on every hand is the therapeutic advice to take milk slowly, "sip it, chew it, take it from a spoon," any way to keep it from forming too large curds in the stomach by too rapid ingestion. We are still daily reminded of it when we are careful not to let the baby take his bottle too quickly. Four different trials were made to determine this point. In each case the milk was sipped slowly over a period of 30 to 45 minutes and then the attempt was made to return it 30 minutes later. In every experiment the result was the same, clear whey only was returned with at most one or two small curds. Only one explanation was possible, that the curds were so large that they could not be returned as they were in every other experiment except one in which after two hours the curd was so large that it lodged for a time in the esophagus and resisted all further efforts to deliver it. That this explanation was correct was further shown by the fact that five hours later a considerable mass of curds could be returned. The result can easily be duplicated *in vitro* by pouring simultaneously milk and rennin slowly over a period of 45 minutes into a beaker under constant stirring. Quite regularly all of the curd will be found in one to four or five large masses, that would be too large to return from the stomach. The explanation is very simple and very convincing as one watches the process in the beaker. Curds coalesce rapidly in proportion as they are fresh. When milk is sipped, or added gradually to the beaker, nascent curds are formed constantly, and attach themselves to the larger masses that have formed till finally only one, or a few very large balls are left. As far as the prevention of large curd formation alone is concerned it can safely be said that milk should be gulped, not sipped!

Modification of Cow's Milk Casein in Infant Feeding.—Nearly all of our methods of adapting cow's milk to the baby result, intentionally or otherwise, in producing a finer curd in the stomach than would be obtained if simple raw whole milk were used. All of these methods in common use have been investigated by the above method on the same individual. As a result it is possible to give a definite account and a concrete picture of what really occurs that was not possible hitherto.

1. *Dilution with Water.*—Dilution with water at first used purely empirically to reduce "the strength of the milk," and later to make the quantity of casein approximate more nearly that of human milk is at once the oldest, the most universal, and one of the most effective procedures we have to modify not only the amount of the casein but also the nature of the resulting curd. If raw whole milk is diluted with an equal amount of water the resulting curds in the stomach "are much smaller, softer and more friable than with undiluted milk . . . they are peculiarly thin, often membrane- or ribbon-like, and all seem spongy or porous. There is also a large amount of crumbly detritus." With still greater dilution all of these differences were accentuated,

though hardly enough to warrant the assertion that cow's milk with its casein reduced to 0.5 per cent. as in human milk would yield a curd like that of human milk. The softness, thinness, and porosity of the curd of diluted milk are doubtless at least as important factors in the greater digestibility of diluted milk as is the reduction in size of the curd. All of these factors materially lessen the distance that purely peripheral digestion must travel.

2. *Dilution with Cereal Waters.*—It has long been known empirically that cereal waters such as barley, or oat meal water, have an even more decided effect on curd inhibition than has dilution with water alone. Cereal waters when properly made have a distinctly slimy consistency due to their colloidal nature. Their action is apparently not so much to lessen coagulation as to prevent the subsequent agglutination of smaller curds into larger masses. The colloid substance probably acts much as mucus does in the stomach by coating the curds. When a quart of equal parts of whole milk and of barley water was taken and the stomach contents returned after 30 minutes "the curds were very small like very fine gravel, of peculiarly uniform size, with only an occasional curd as large as a small pea. They were very soft, shreddy or scraggly, porous and of greenish gray color. . . . They were very much finer than those in which an equal or treble amount of water was used as a diluent . . . in fact they in no way resembled them." There was no appreciable increase in the size of the curds after one hour as compared with 30 minutes and nearly all of the curds then presented "a finely flocculent homogenous, somewhat gelatinous mass that resembled cooked starch in which individual curds could hardly be made out." When one sees these uniformly fine, peculiarly porous, soft curds, and the conditions at the end of an hour as compared with those of a like dilution with water alone one can easily understand why barley water has held its great popularity for so many years. The same properties here noted doubtless exist in all cereals and also in pure starch; but *do not exist in any of the soluble carbohydrates, i.e., in the sugars and the dextrins.*[9]

It has been stated that the minimum amount of starch that will produce the optimum result is 0.75 per cent.; that larger amounts have no greater effect. Inasmuch as this was not tried in the stomach itself the deduction is of little value. Practical experience with the use of barley water as ordinarily made and my own stomach experiments would lead me to think that the application of finer mathematic calculations is at least superfluous. Starch is so uniformly well tolerated in such small amounts at any age that numerical dosage hardly seems necessary.

3. *Peptonization.*—Peptonization changes the casein ultimately into peptones and these are no longer coagulable. In the period when all of our fears in infant feeding centered about the casein peptonization was a frequent and certainly most effective weapon in combating that danger. With more sharply defined knowledge of the real and assumed dangers of casein indigestion, and of other simpler methods

of meeting that difficulty peptonization has practically fallen into complete disuse except in rectal feeding.

4. *Precoagulation of the Milk.*—Milk that has once been coagulated, whether by acids as in buttermilk; or by rennin, as in the use of pegnin, or chymogen; that has then been actively stirred, shaken, or beaten up, and allowed to stand for a time; shows very little tendency to recoagulate in the stomach.[7] In the case of the sour milk there was in my experiments no appreciable change;[9] in the case of the milk coagulated with rennin the curds were "quite uniform in size like a coarse sand or a very fine gravel. They were peculiarly soft and mushy, irregular and scraggly looking, having no resemblance to raw milk curds." Both of these expedients are used in *Eiweiss milch* which is a combination of finely divided curd of sweet milk that has been coagulated by rennin, and of buttermilk. When tested in the stomach there was found "as in buttermilk alone, practically no tendency to the reformation of curds in the stomach." These three preparations are all so extensively used in infant feeding that they will be considered more extensively later.

5. *Boiling of the Milk.*—Boiling very materially inhibits curd formation in the stomach. Instead of the tough, massive curds, 3 and 4 in. long, that are formed when whole raw milk is taken, we have a very different curd when milk is boiled. When a pint of milk boiled five minutes was returned from the stomach after 30 minutes "the curds were very uniform in size. The great majority of them were the size of a small pea, a few were a little larger, and the rest varied all the way down to fine sand. Fully as striking was the difference in consistency. Instead of hard and rubbery, they were soft, friable, scraggly, but rounded off and resembled very closely a finely broken up good custard."[9] As in the case of raw milk the curds of boiled milk increased to a maximum of size and hardness after two hours, but they were no larger than a pea or a very small hazelnut. The longer the milk was boiled the greater the effect on coagulation. When boiled milk was diluted with equal parts of water the resulting curds were "fine, soft, about like coarse sand." Pasteurization has very little effect on coagulation.

6. *The Use of Evaporated, Dried, or Condensed Milk.*—It has long been known that condensed milk probably occupies a place next to human milk as the most universally easily digested infant food. Probably one of the reasons at least lies in the casein. When diluted (1 : 10) and returned from the stomach after 30 minutes "the curd was an almost imperceptible, very minute, soft, flocculent, precipitate that separated from a nearly equal amount of almost clear water, and settled to the bottom as a fine, white, sand-like sediment."[9] Even when used in 1:4 dilution which would represent nearly 2 per cent. of casein, and returned in one hour "the curds were soft and flocculent . . . . characteristically uniform in size and like a moderately coarse sand, but not a single curd was larger than a large grain of sand." Horlick's Malted Milk and Nestle's Food, both containing dried, or con-

densed milk as a base, and a dried milk, Dryco, all gave identical, or even more striking reactions. In general these milks seem to represent as far as the coagulation of the casein is concerned the third step, of a series of which raw milk, and boiled milk respectively form the first and second steps.

7. *The Use of Alkalies.*—Alkalies and certain salts have been used extensively in milk modification, at first empirically and more recently for their demonstrated effect on the coagulation of the casein. Those most commonly used are lime-water, sodium citrate, sodium bicarbonate, potassium bicarbonate, and milk of magnesia. All of these were tested by the above method in the stomach itself and the results in each case fully warrant the assertion that they all have a marked effect in preventing the coagulation of the milk, though the method of action varies.

Lime-water is the oldest and still most used representative of this class of milk modifiers. At first used empirically to "sweeten the stomach" in vomiting babies; to "overcome the acidity of cow's milk;" to furnish "lime salts as a preventative of rickets;" it has more recently been used with the idea of inhibiting curd formation. There is ample evidence both clinically and experimentally that it does this very effectively. When 5 per cent. of lime-water was added to 30 oz. of milk and returned from the stomach in 30 minutes "the curds were about midway in size between those of raw and of boiled milk but they were peculiarly thin, scraggly and band like, and were so porous, soft and friable that they could hardly be picked up without breaking." When 10 per cent. of lime-water was used the curds were still smaller and even more ribbon like or flaky, more porous, soft and friable than when 5 per cent. was used. The returning curds were reported as "the softest" in any experiment and the stomach contents were said to have "a very sweet taste" that was present in no other experiment. The curd of milk to which lime-water was added seemed different from any other curd, and that chiefly in the extreme softness and friability, the greater porosity, and even more strikingly the thin ribbon-like nature of nearly all the curds. While 5 per cent., or 1 oz. to every 20 oz. of milk, is the amount commonly used there is no objection to a very much larger amount, and the effect upon the curd is directly proportional to the amount used.

Sodium Citrate.—While sodium citrate is not strictly an alkali and its action is probably quite different from that of the alkalies clinically its effect is indistinguishable from that of the latter. It has been used most extensively in England and to a large extent in this country. To get the maximum effect it is customary to add 2 gr. to each ounce of milk in the mixture, while lesser amounts such as 1 gr. to each ounce is often used for a less decided effect. In my experiments 1 gr. gave after 30 minutes "a soft curd in size and consistency about midway between those of raw and of boiled milk." When 2 gr. were used "there was practically no separation of curds and whey" after 30 minutes. "The milk was simply thickened resembling a good cream soup, with no distinguishable curd formation, except scattered small, soft floccules." At the end of two hours the stomach contents still looked "like a pale thickened milk," in which was contained about a dessert spoonful of harder curds, "most of them like fine gravel, two curds the size of a bean, and one bizarre aggregation was ½ in. long, 1 in. wide, and ¼ in. thick." Thus while sodium citrate has a marked inhibiting action at first, there is nevertheless present the usual tendency for such curds as are formed to get larger for a period of two hours.

Sodium Bicarbonate.—When sodium bicarbonate was used the effect on coagulation was indistinguishable from that of sodium citrate. When 2 gr. to the ounce were used, however, "the mixture was so nauseating and so hard to take on account of the violent eructations of gas and the disagreeable taste of the 'soda' that it was impossible to take all of it. . . . No other experiment was accompanied by anything approximating the discomfort and the disagreeableness of this one." No tests were made with smaller amounts as in the case of sodium citrate. Because of these features sodium bicarbonate is rarely used in larger amounts. Sodium citrate has none of these objections.

Potassium Salts.—The potassium salts are rarely used for their action on coagulation. The presence of potassium bicarbonate in Mellin's Food (2.5 per cent.) doubtless accounts for the finer curd produced than when other dextrin-maltose preparations are used. Mead's Dextri-Maltose No. 3 now contains potassium carbonate, as do also the malt extracts that are used for making malt soup. In general they have probably the same action as the sodium salts.

Milk of Magnesia.—While milk of magnesia is commonly used for its laxative action, it is also a powerful antacid and milk coagulation inhibitor. While a teaspoonful of milk of magnesia (Phillips') contains only about 3 gr. of magnesium oxide in the form of a hydroxide this amount had fully as decided an effect on the coagulation of milk in my experiments as did 30 gr. of sodium citrate, in fact the two results were indistinguishable after 30 minutes and after two hours. The added laxative properties of milk of magnesia would seem to make it a most favorable alkali to use in infant feeding because of the frequency of constipation in the bottle fed baby. The baby that is constipated, however, rarely requires any casein modifier. For this reason milk of magnesia is commonly used only as a laxative and its other properties are disregarded.

The mode of action of these alkalies and salts has been the subject of much discussion and is apparently not the same for all of them. In the case of alkalies like magnesium hydroxide (milk of magnesia), calcium hydroxide (lime-water), and sodium and potassium bicarbonate, the predominating action is doubtless that of neutralization of the hydrochloric acid of the gastric juice. Rennin acts only in an acid or neutral medium. The neutralization of other acids in the stomach, as well as of the lactic acid of the milk itself, probably also plays a part. Sodium citrate on the other hand is at most but a very weak antacid. While it probably reacts to some extent with the hydrochloric acid forming citric acid which is a weaker acid than hydrochloric acid and thus reduces the total acidity, this action would be totally inadequate, as the stomach contents would still be acid. The explanation offered by Bosworth and Van Slyke[12] would seem to meet all objections and is as follows: Casein is present in milk in combination with calcium as calcium caseinate (either tetra-, or tri-calcium caseinate) and has, therefore, the characteristic properties of an acid in that it combines with metals or bases to form caseinates. When this calcium caseinate is acted upon by rennin each molecule is split up into two molecules of calcium paracaseinate. This is less soluble than calcium caseinate and is, therefore, precipitated, that is the milk curdles. When sodium citrate is added to milk there is a reaction with the calcium caseinate leading to the formation of sodium caseinate (or calcium-sodium caseinate) and calcium citrate. Sodium caseinate when acted upon by rennin splits up into two molecules of sodium paracaseinate which is very soluble, i.e., the milk does not curdle. They found that when 1.7 gr. of sodium citrate was added to each ounce of milk the point was reached where coagulation no longer took place, i.e., the calcium caseinate of normal milk had been changed to sodium caseinate, or the double salt calcium-sodium caseinate and the calcium-sodium paracaseinate formed when this is acted upon by rennin is very soluble. When a lesser amount of sodium citrate is added a lesser amount of calcium-sodium paracaseinate is formed when acted upon by rennin, that is coagulation is delayed or prevented to that extent.

It is evident from the above why such different amounts of the various alkalies and salts are necessary to produce the same clinical result. Hydroxides such as magnesium hydroxide and to a less degree calcium hydroxides have a much stronger immediate affinity for acids than have the salts such as the carbonates and the citrates. A very much smaller amount of magnesium hydroxide is necessary to neutralize the hydrochloric acid of the stomach as compared with the amount of sodium citrate than is necessary to combine with the very much larger amount of calcium salts in the casein of the milk to produce the same result. A teaspoonful of milk of magnesia containing 3 gr. of magnesium hydroxide has apparently about the same effect on coagulation as 30 gr. of sodium citrate or about 8 oz. of lime-water (6.5 gr. of CaOH), or about 15 gr. of sodium bicarbonate. In practice 5 to 10 per cent. of lime-water, or 1 to 2 gr. of sodium citrate to each ounce of milk are the proportions commonly used.

The Salts.—As we have seen cow's milk contains about 0.70 per cent. of ash, human milk about 0.21 per cent., i.e., is roughly three times as much. In a general way all the salts are represented in greater amount in cow's milk than in human milk and the common dilutions used in feeding young babies roughly equalize this natural inequality. To this there are several exceptions and these are the only ones that are of interest in discussing the modification and adaptation of the food element of cow's milk in infant feeding. The calcium salts as we would expect from their combination with the casein are very much more abundant in cow's milk than in human milk, while the potassium and iron salts are found in relatively smaller proportion in cow's milk. While, too cow's milk contains more phosphorus than does human milk less of it is in organic combination than is the case in human milk and this may be of significance that is not yet measureable.

The rôle that the different salts play in the pathology of infancy is not yet clearly defined and relatively little can be done to modify or adapt them to the infant. In the first place it is a relatively new field and while an enormous amount of work has been done precision and agreement are still wanting for practical purposes. In the second place the utilization by the baby of the salt of cow's milk and of human milk seems widely different. The breast fed baby is said to absorb 80 per cent. of the total salts of its food as compared with 43 to 78 per cent. of the artificially fed baby.[13] In the breast fed baby furthermore 40 to 50 per cent. of the salts are retained while in the artificially fed baby the retention may be all the way from this amount down to nothing depending upon the nutritional state of the infant which is far more uncertain than in the breast fed baby. Again salts are excreted not only in the urine but also into the intestinal tract and this makes it difficult to estimate the total absorption and retention. The fact that more of the salts of breast milk are in organic combination, as for example, the phosphorus, than is the case with cow's milk may account for the greater utilization in the former. Lastly, the absorption and retention probably depend to a considerable extent upon the associated fats, carbohydrates, and protein, and these are not the same in cow's milk as in human milk. Thus according to Hoobler[13] the retention of calcium and of phosphorus is greater in food rich in fat and of magnesium in a food low in fat.

The *iron* of both cow's milk and human milk is so small in amount (0.00004 to 0.00007 per cent. and 0.0001 to 0.0004 per cent. respectively)[6] that it is inadequate alone to cover the child's need. While the figures given as the results of different investigations vary very widely it now seems probable that cow's milk contains less than human milk. Even this amount (less than 0.01 gr. in a quart of milk) is materially lessened in practice because of the dilutions commonly used in early infancy and also because of the poorer utilization of the iron of cow's milk as compared with that of human milk. It is generally accepted that this amount is adequate for the infant only because a large amount of iron has been stored in the liver that is utilized during the first five or six months. After this period the infant must receive more iron than it gets from milk alone, and especially from cow's milk, or it will become pale, soft, and inactive as is well known clinically. This deficiency is readily made up by giving cereals, broths, vegetables, etc., from the fifth month on. If for some reason that is hardly conceivable, this is not feasible, then small amounts of iron would seem indicated.

The *calcium* of cow's milk is approximately four times as abundant as it is in human milk. Even with the maximum dilutions used in infant feeding there is always more than in human milk. Human milk contains so small an amount that there would at times seem danger of an inadequate amount to cover the infant needs. This is all the more interesting because rickets, in which there is a faulty calcium metabolism, is very rare in the breast fed baby and very common in the cow's milk fed baby. We have again to do with a difference in the utilizability of a salt when found in cow's milk and when found in human milk. This will be discussed fully under rickets. The infant that is on the breast, or on a moderate amount of cow's milk receives enough calcium. It is, therefore, useless to add a calcium salt to the food except where these requirements are for some reason not met. In certain conditions such as tetany, or spasmophilia, that are associated with rickets and in which it has been demonstrated that the calcium content of the blood is materially diminished the administration of calcium salts such as calcium chlorid is indicated and is therapeutically effective. Both cod-liver oil and phosphorus and especially the combination of the two have a marked effect on calcium utilization, and while the exact mode of action is not clearly determined, clinically there is no doubt that they have a well-nigh specific action in the treatment of rickets, and of associated conditions such as spasmophilia. It is also a well known fact both clinically and experimentally that there is an interrelation between calcium utilization and some of the other food elements. Thus the carbohydrates favor the utilization of calcium and the fat of cow's milk if in excess has an opposite effect. This is clinically best illustrated in the fact that the infant in the second semester of life that gets an excess of milk and has soap stools due to fat overfeeding nearly always becomes seriously rachitic unless its food is changed by materially lessening the milk, i.e., the fat, and correspondingly increasing the carbohydrates, either by addition of sugar and starch to the bottle or by the additional feeding of cereals, fruit juices, vegetables, etc. There is here not so much an antagonism between the calcium and the fat, for in other cases the fat favors calcium retention, as that in this condition the calcium is diverted to form soap stools, that is salts of the fatty acids that result from feeding an excess of cow's milk fat. In this condition the more calcium, i.e. the more milk, given the less it is utilized and the more surely will rickets manifest itself. In general then, the adaptation of cow's milk calcium to the infant consists not in modifying it, nor yet in increasing or diminishing the amount except in most rare instances, but in safeguarding its utilization by bearing in mind its interrelation with the carbohydrates, the fat of cow's milk, and with cod-liver oil and phosphorus.

The *phosphorus* is used chiefly in the nervous system, and in the formation of bone where it occurs in combination with calcium. For obvious reasons it is represented in about the same excess in cow's milk as is the calcium. Because of its association with calcium its metabolism runs a more or less parallel course. The fact that a lesser amount of phosphorus is in organic combination (nucleon, lecithin) in cow's milk than in human milk may cause it to be less well utilized, and this may be a factor in the greater occurrence with the former of rickets, and spasmophilia, where there is a disturbance of calcium, and possibly of phosphorus metabolism. Even in the maximum dilutions of milk the artificially fed baby is given more phosphorus than in its natural food. The favorable action of phosphorus and cod-liver oil in rickets, and associated nervous conditions (spasmophilia) points to the conclusion that in these conditions the utilization of the phosphorus of cow's milk suffers with that of calcium in the infant's economy.

The *potassium salts* are about three times as abundant in cow's milk as in human milk, but relatively less so than most of the other salts. There is again evidence that these salts, too, are utilized less fully with cow's milk as compared with breast-milk feeding. Metabolism experiments would, however, seem to indicate that the retention is determined rather by the need than the amount taken and that even in the common milk dilutions the amount of potassium retained in artificial feeding is always enough to avoid a negative balance.[6] The *relatively* small potassium content of cow's milk as compared with that of human milk, and its possible relation to the striking difference in behavior between the two milks when fed to an infant, has been a factor in various attempts that have been made by Friedlander and Schloss to make a milk that would act like human milk. Among other things these consist in reducing the whey salts and adding potassium chloride. (See Whey Reduced and Whey Adapted Milk.) From what has been said there is at least doubt as to the value of adding the potassium salt.

Reduction of All Salts.—More recently a reduction of the excessive salts of cow's milk, together however with a reduction of the milk sugar as well, has been brought forward by Finkelstein as a therapeutic measure in digestive disturbances with fermentation (Eiweissmilch). The reduction of the salts is doubtless, however, only a minor factor in the striking efficiency of this therapeutic food.

In general one may say that the role of the salts in the problem of artificial feeding is now receiving the most intensive attention by investigators and it seems probable that they will be found to have a greater practical significance than has been assumed in the past.

The Water.—Water is more immediately and continuously essential to life than is food. There is furthermore an optimum concentration of body fluids for all processes of digestion and metabolism that is quickly impaired if the intake is seriously altered. In the breast fed baby there is naturally a normal relationship of the water intake to that of the other food elements and we are little concerned about the water intake, *per se*, provided the baby is thriving. Likewise with a food so rich in water as are both breast milk and cow's milk (87 + per cent.) it would seem quite unnecessary to give the large additional amounts of water that are so commonly given. In general a baby in the first few months of life contains about 70 per cent. of water and requires about 140 to 150 gm. per kg. (2 to 2½ oz. per pound) of water daily.[6] This gradually diminishes as the child gets older, and the adult requires relatively only about one-fourth as much. The bottle fed baby usually gets more water than the breast fed baby because of the greater dilution of the milk, and hence, the necessity for a larger amount. In the healthy baby who is bottle fed there is relatively little danger of giving so little water in the food that the baby's needs are not covered. At a time when both food and water are required and thirst and hunger merge the baby would probably not fail to make its wants known and would get more of one or the other. There is generally more likelihood that the baby will get more than is necessary especially when, as so often happens, any cry is interpreted as hunger and the baby gets up to 40 to 60 oz. of food a day. Such amounts are never necessary and can serve no useful purpose, and may even be harmful. In hot weather more water is needed, for obvious reasons. In normal conditions it is always well to give some water daily both to supply its needs if it seems to want it, and also in the breast fed baby to accustom it to the bottle.

In the sick baby the water intake requires more careful attention. Such a baby will commonly refuse nearly all food, and is only given such amounts of water alone as it was getting, or relatively little more, because the amount of water that it formerly got in its food is disregarded. Its need for water is the same as before and should equal it in amount. In the febrile child, and in the child with diarrhea the demand and the need for water are above normal and must be satisfied, not curtailed as is done only too often. The weak, semi-conscious, even comatose, baby and the baby that vomits everything urgently demand such special methods of safeguarding the water intake as naturally suggest themselves to the experienced physician. In certain pathological conditions the diminished blood volume, and the increased blood and body fluid concentration, determine the fatal termination rather than the primary condition itself.

There is again an inter-relation between the water and other food elements, notably the salts and the carbohydrates. The action of salt on water intake and in nephritis on water retention is a matter of common knowledge. Carbohydrates, and especially starch, favor water retention. Infants fed almost exclusively on starchy foods may show normal weight and unusually firm tissue turgor, due to excessive water content (Mehlnährschaden, Czerny, Keller).

Historical Résumé of the Development of our Knowledge of the Role of the Different Food Elements in the Digestion of Cow's Milk.— In the development of our knowledge of artificial feeding one after the other of the different food elements has been brought into prominence as the chief factor that makes for poorer digestion and utilization of cow's milk than of human milk by the infant. In his monograph, published in 1865, Liebig describes his Malt Soup, and speaks only of the necessity of having the "blood producing" (protein) and the heat producing components (fat and milk sugar) in proper proportions in an artificial food. With clearer knowledge of the composition and digestion of milk it was natural that the casein should first be accused as the chief offender. In 1869 Biedert published the first of a long series of papers dealing with the differences between human and cow's milk. As a result of coagulation experiments with rennin on the two milks and from clinical observation, and especially the obervation that babies with digestive disturbances passed hard curds in their stools which he interpreted as undigested casein, he considered the casein as the food element that distinguished cow's milk from human milk. He attempted to make a food like human milk the essential factor being the reduction of the casein to approximately that of human milk. The same views were held and the same attempt made by Rotch in this country a few years later.

Biedert's views concerning the digestibility of the casein, and the significance of the curds of the stools held sway for a long time. Gradually doubts arose as to both the difficulty of digestion of the casein, and the casein origin of the curds of the stools. Soon practically all leading German pediatricians were agreed that the casein was not difficult to digest, that metabolism experiments demonstrated that the casein of cow's milk was utilized as fully as that of human milk, and furthermore that the so-called "casein curds" contained no casein, but were made up of fat derivatives.

In 1906–9 appeared the epoch making work of Czerny and Keller[14] in which the role of each food element was treated in relation to the nutritional disturbances of the artificially fed baby. The casein was absolved of all blame and the "casein curds" were shown to be fat in origin. The carbohydrates and the salts were assigned very minor parts. In the *fat* they found the chief difference between cow's milk and human milk, in fact they designated the disturbance due to overfeeding with fat, *Milch nährschaden*, i.e. a nutritional disorder due to milk; or, obversely, they maintained that a nutritional disturbance due to milk was due to the fat of the milk. This view was quite universally accepted in Germany, and to a large extent in this country.

More recently still (1910) Finkelstein and his school have approached the whole subject of the pathogenesis of the nutritional disorders from a different viewpoint that is discussed elsewhere. For our present purposes it is enough to say that they have raised the sugar to the first position in the genesis of the more serious disorders of nutrition in infancy, and have associated with this the whey salts. The efficacy of *Eiweiss milch*, in which nearly all of the milk sugar and half of the whey salts are removed, in all digestive disorders with fermentation would seem to speak for the soundness of this view. It is certainly a matter of clinical experience that no food element must be used more cautiously than the sugar, and especially milksugar in the presence of diarrhea. The position of the salts as yet cannot be properly evaluated.

Thus each of the food elements has had its turn. Only the water remains and latterly we are hearing much about *anhydremia* as the important factor in the severe intoxications with diarrhea and vomiting.

This peculiar succession of ever varying ideas seems inexplicable except for two reasons. Some of the differences are apparent rather than real; i.e., differences of viewpoint rather than of fact. In the second place certain other developments ran parallel with those outlined and explain certain of these discrepancies. Probably the greatest difference of opinion that has existed in all of these developments has centered about the digestibility of the casein. To be held the greatest offender in one decade and to be declared wholly innocent in the next could not well happen without much discussion, and moreover for some good reason. In these discussions the main attention focussed ultimately upon the "casein curds" of the stools. Again it was hard to see why in one decade they should have been "weiss bräunlich-gelbliche Brocken und Knollen" (Biedert)[1] and manifestly due to casein, and a little later "small, soft, whitish or greenish"[14] equally evidently derived from the fat. In this country, furthermore, especially in the East, the views of Czerny and Keller concerning fat did not seem to meet with approval, nor did the views of Biedert and Rotch concerning the difficulty of digestion of the casein meet with disapproval, in fact it long remained as the keystone of Percentage Feeding. The whole matter finally came to a focus in a discussion between Talbot of Boston on the one hand and Meyer and Leopold of the Finkelstein School on the other hand. Talbot[15] in a series of papers had shown quite incontestably that there were two kinds of curds, a hard curd and a soft curd; that the hard curd was made up chiefly of casein, and the soft curd as evidently of fat derivatives. South worth and Schloss,[16] too, had drawn attention to this difference and it was a matter of common observation in this country. Meyer and Leopold[17] categorically denied all of these assertions basing their conclusions on chemical and metabolic investigations, in spite of the fact that Talbot advanced incontravertible evidence based on chemical and even specific precipitation reactions with these hard curds and the serum of sensitized rabbits. The discussion ended in a draw—both sides accepting the decision! The simple explanation of this difference of opinion was given by the work of Ibrahim[18] and myself,[19] which appeared independently and practically simultaneously. We had both observed that hard curds occurred only when raw milk was given, and disappeared when the milk was boiled. In a series of babies I had made and unmade hard stool curds at will by giving alternately raw and boiled milk. German clinicians for many years have used only boiled milk. Talbot and nearly all American clinicians were using raw milk. Meyer had never seen a hard curd until he made them after this discovery by feeding some of his own babies raw milk.

The descriptions that Biedert gives of "casein curds" "whitish-browish-yellowish nodular masses" leaves no doubt that he fed *raw* milk and saw hard curds. The later observers all used *boiled* milk and so were perfectly correct when they maintained that the curds they observed and examined were fat curds, never casein curds.

The views of Czerny and Keller and of nearly all more recent German pediatricians concerning the harmlessness of the casein and the harmfulness of the fat, as compared with the views of say the Boston School which clung to the harmfulness of the casein, and found little evidence of the harmfulness of the fat in the Czerny-Keller sense, can also be harmonized on the same hypothesis. In further investigations on the coagulation of raw and of boiled milk (see "Boiled Milk") in the stomach itself I[9] found that the reason for the occurrence of the "hard curds" with raw milk lay in the fact that raw cow's milk forms enormously large curds in the stomach that under certain conditions pass clear through the digestive tract undigested and appear as "hard curds" in the stools. That they do not occur when boiled milk is given because boiled cow's milk makes a relatively soft and very small curd in the stomach that is much more quickly digested by the digestive juices that act only on the surface and can naturally digest a very short distance more quickly than a much longer distance. When feeding boiled milk one can give much larger amounts and of greater concentration without producing digestive disturbances than if one gives raw milk, as those of us well know who have had extensive experience with both. Raw milk when given in sufficient amount, i.e., excessive amount for that particular case, and of sufficient concentration will practically always lead to casein indigestion, often with the presence of casein curds in the stool, long before the characteristic appearance of the fat constipation of Czerny-Keller. With boiled milk casein indigestion is practically abolished when given in therapeutic amounts and when milk is given in excess of an infant's tolerance the normal result is that observed and described by Czerny and Keller as Milchnährschaden. In other words, while we fed raw milk we shared in the fear of the casein and relatively rarely saw fat constipation; since we have used boiled milk we have come to disregard the casein, and we avoid fat constipation only by restricting the amount of milk to a point where this does not appear in serious form and by substituting carbohydrates to an extent necessary to prevent its occurrence.

If these observations are correct we have run the gamut of all the food elements, have even started on the second lap, and find them all involved in the large problem of why cow's milk is harder to digest by the baby than is its natural food. Consciously or unconsciously we are constantly meeting the problem of difficult casein digestion by nearly every device that is current in modifying milk for feeding babies; we know that the fat[7] is probably the hardest food element to digest, that it leaves the stomach more slowly than any other, and that it most easily causes vomiting and indigestion with diarrhea on the one hand and a most obstinate constipation accompanied by a severe metabolic disturbance on the other; that the sugar of milk is the chief factor in the genesis of fermentation—one of the largest items in the nutritional disorders—and that it must be used in cautious dosage and must often be replaced by the other carbohydrates; and, lastly, that the salts are increasingly being given a more important part in the various digestive and metabolic processes, both normal and pathological. At every turn we are reminded, furthermore, of the fact that an individual food element may be harmless and benign when given in a certain medium with certain other food elements; that it can be harmful and destructive when given in combination with other food elements in a different medium.

Intimately interwoven with all this and still wholly imponderable is the part played by bacteria. This will become evident in various portions of the text and especially in the chapter on the nutritional disorders, and in the following paragraphs.

COMPARATIVE BACTERIOLOGY OF HUMAN AND OF COW'S MILK

Countless bacteria accompany all digestion and especially milk digestion. Nearly all of them are harmless, many even beneficial. To draw the line between the harmful and the benign is possible only within certain limits. Furthermore the same organism that at one time is harmless or even beneficial, in a different condition can become pathogenic. The bacteria found in both human and cow's milk must be considered from two angles—those that are present in the milk when drawn and those that are added after it has been drawn and before it reaches the baby.

Human milk is not wholly sterile when it leaves the nipple. In the first milk the staphylococcus aureus is not uncommonly found, occasionally others, that have found their way into the *sinus lactiferus*, or cistern of the nipple, from the outside. They are probably always harmless. When the breast is the seat of a localized inflammation, or suppuration, it is quite possible for the causative organism to escape through the nipple during nursing. Even here there is little evidence that the infant will suffer as the result of the ingestion of pyogenic bacteria especially if it is normal in every way. The possibility of transmission of tuberculosis through the nipple seems remote. Local tuberculous infection is probably a *sine quo non*, and this is very rare and would almost inevitably attract attention to itself. In cases where there would be danger from this source, or where the bacteria might be carried to the gland through the blood stream and there excreted, if this were granted as possible, there would be so much evidence of tuberculosis elsewhere, as is so common in the tuberculous puerperal mother, that it would lead to weaning and so to a removal of the danger. Only in rare instances have pathogenic organisms, such as that of typhoid, where the mother was infected, found their way to the baby through the milk.

External contamination of breast milk in direct nursing could come only from unclean nipples and from the infant's mouth. The latter is negligible, and the former with the most ordinary cleanliness is nearly so. Touching the nipple with contaminated fingers would be a most likely source of trouble but seems rare.

In the cases of *cow's milk* where the udder is normal there is the same condition as in breast milk. There is, however, a further danger that the milk can become contaminated with pathogenic organisms even *before it leaves the udder.* A diseased condition of the udder would be less apt to concern a milkman than a diseased breast would the mother, and we have here a real source of danger. The two organisms that need be considered are the streptococcus, that has been held responsible for epidemics of streptococcus sore throat, and the tubercle bacillus, which has frequently been found in milk from tuberculous udders. While this is probably the most frequent source of contamination of cow's milk with tubercle bacilli there is also danger from the outside.

In cow's milk the danger from *external contamination* with pathogenic organisms is a very real one. Among the diseases that have been traced directly to such milk are tuberculosis, typhoid, dysentery of the various types, Asiatic cholera, streptococcus sore throat and enteritis, diphtheria, scarlet fever, anthrax, actinomycosis, foot and mouth disease, and Malta fever. A great variety of modes of infecting the milk from the time it leaves the cow till it reaches the baby naturally suggest themselves. We have here an excellent culture material handed for 24 to 48 hours from one individual to another none of whom knows or cares anything about bacteriology. During all this time it is exposed to one or another source of contamination—dust from the air, dirt from the hands of the milker and others, dirt and dried feces from the sides, udder and tail of the cow, contact with diseased individuals and "carriers" of disease, unclean utensils, etc. Fortunately nearly all the organisms that thus enter the milk are harmless, consisting in part of wholly negligible organisms, partly of such organisms as the colon bacillus, and predominatingly of lactic acid producing bacteria that cause the milk to sour and coagulate, and of which some one hundred varieties have been isolated. These acid producing organisms are probably not only harmless but even beneficial in that they inhibit the growth of other germs that might be harmful. Whether they are themselves ever harmful to a baby lacks clear evidence, and leaves one in doubt in the presence of the baby that "is upset because its milk was sour." It is interesting in this connection that natural buttermilk made from soured milk has been used extensively with signal success in the treatment of digestive and fermentative disorders in infancy. It is also claimed that if these organisms are killed and the milk then allowed to spoil it will decompose rather than sour normally. This view certainly seems rational, but the evidence in favor of this as a factor in the pathology of infant feeding is so meager that few of us have ever encountered it.

The tubercle bacillus is, after all is said and done, the one organism that concerns us most in this connection. The percentage of cows that are infected with tuberculosis is staggering, especially when we consider that in the commercial production of milk the vast majority of these are controlled neither clinically nor with the tuberculin reaction. The disease needs neither to be far advanced so that it is grossly recognizable, nor yet to involve the udder to be a source of danger. A small open focus in the lung leads to the presence of organisms that are carried or coughed up into the mouth and swallowed. They leave through the feces and are easily carried to the milk by paths that are well known to anyone who has ever milked or has seen someone else do it. The milkman who has a cough and is not particular as to his aim can easily add the human to the bovine variety. He would likewise pay little attention to a lump in the teat or the udder. How great the danger of human infection with tuberculosis, human or bovine, from milk, and just what the quantitative incidence of human and bovine tuberculosis may be in general and at various ages still requires sharper determination, but all the evidence points to the possibility of a very real and a very extensive danger. Under any circumstances the problem has a vital significance to the pediatrician, for if the danger is real then it is most acute in infancy. Infants take more milk and the younger the individual the greater the danger of infection with tuberculosis and especially of the bovine type. So long as Von Behring's hypothesis that tuberculosis is commonly acquired in infancy and becomes active only when conditions are favorable has not been disproven so long one can only choose in practice to act as if this maximum danger really had been demonstrated.

Confronted with such a problem two alternatives present themselves. Milk must either be so safe guarded from before the time it leaves the cow until it reaches the baby that all of these dangers are reduced to a minimum, or else the milk, in addition to all reasonable care in its production and handling, must be subjected to heat or some other agent that will destroy or at least inhibit the growth of the organisms that are inevitably present. This leads us, on the one hand to certified milk, and on the other to heated milk as the only feasible practicable alternatives.

Certified Milk.—Certified milk is discussed in detail in Chapter XXI. In this connection the subject will be treated only briefly from the present viewpoint. By certified milk is meant a milk produced and handled in accordance with certain minimum requirements laid down by a milk commission appointed usually by a city or a medical society, evidence of compliance with which requirements on the part of a milk producer leads to a certification by this commission that his milk is produced and handled in accordance with these requirements and can be sold so long as he meets these requirements as "certified milk." The idea was fathered by Dr. Henry Coit, of Newark, N. J., to whom the greatest credit is due. The purpose then as now was to produce as unexceptionable a milk as possible for babies, one that could be used raw with safety.

Briefly the requirements that are laid down for the production and handling of certified milk are the following:

1. Utmost cleanliness about milking, stables, cows, udders, hands, pails, bottles, and other utensils with which the milk comes in contact from the time it leaves the producer till it reaches the consumer.
2. Immediate cooling of the milk to a low point and keeping it constantly at a low point by the use of ice until it reaches the consumer.
3. The milk should be transported from the producer to the consumer in the shortest time possible.
4. The cows should be healthy, should be kept hygienically, and tuberculosis should further be excluded as nearly as possible by the application of the tuberculin test as often as necessary.
5. The milk commission, or someone appointed by them, should see that all requirements are at all times complied with.

When milk is thus produced and kept the bacterial dangers sink to a minimum, if not to the vanishing point. That such milk is preferable to ordinary milk both bacteriologically and esthetically needs only to be stated, and this has led to a general improvement of all milk production and handling. Some misapprehension exists among the laity and even among some physicians as to the nature and significance of certified milk. Uninformed and poor mothers, especially, with whom certified milk is a sacrifice because of the necessarily greater price, feel when they make the sacrifice as if they were getting a sort of glorified infant food comparable in a way to mother's milk. Certified milk is, of course, simply a clean, fresh, relatively germ-free, raw milk that requires identically the same treatment in being converted into a food for the baby as any other milk except that it is safer to use raw.

Heated Milk: Boiled, Sterilized, or Pasteurized.—Few questions pertaining to artificial feeding have been discussed more furiously, and less fruitfully, for many years than that of whether milk should be given to babies raw, or boiled, sterilized, or pasteurized. While the general problem of heated milk is discussed in detail elsewhere it would seem to require clinical discussion here.

Naturally, at first, milk was given to babies as it came from the cow, or the goat. Mothers soon learned that milk kept better and was more acceptable as a baby food when it was boiled. We have seen how Metlinger[3] in 1473 advised the use of "goat's milk boiled with one-fourth part of water." The advent of bacteriology furnished a reason. Infantile diarrheas were assumed to be due to infections, and cow's milk teemed with countless bacteria. Sterilization of the milk naturally followed and that in a manner not unlike the methods in use by bacteriologists. Many of us can remember the time when the baby's "sterilizer" occupied a position in the household not unlike that of the family altar. With an increasing knowledge of a certain class of nutritional disorders, at the head of which stands infantile scurvy, in which apparently some vital principle necessary to perfect nutrition was destroyed, or absent, in the food on which it was produced, came the first doubts as to the unquestioned virtues of sterilization. It was found that in nearly every case some proprietary food was used, or some food that was old or stale, that it occurred relatively frequently on condensed milk, only occasionally on sterilized, or cooked milk, almost never on raw milk, and practically *never* on mother's milk. It was natural to conclude in practice at least, though the evidence was uncertain, that the essential factor in the production of scurvy was the destruction of the causative vital principle by heat and that other yet unknown dangers lurked in this at best unnatural practice. Besides mother's milk was raw, all young mammals drank only raw milk. The change to raw cow's milk in the routine of infant feeding naturally followed, and with it the demand for a milk that could safely be fed raw, i.e., certified milk. Recently again it has become evident that the question of whether milk should be given raw or heated is not wholly a bacteriological, nor a biological problem, but also a physiological problem. Boiled cow's milk for reasons that have been detailed is unquestionably much easier to digest than raw cow's milk.[7,9] With the problem of scurvy still unsettled, and having at hand always a simple and absolutely sure preventative of scurvy in the use of small doses of orange juice, many of us are boiling certified milk for babies because it is easier to feed them than raw milk. The sterilizer with its ritual will probably never come back, but the sauce pan threatens to take its place.

As a result of all these changes in viewpoint there has naturally grown up in this country, and in this country alone, a strange transitional medley of ideas of different men who have followed, or have not followed, these changes with an unequal pace. Some of us have used boiled milk for many years and are still looking for the first explosion. With others raw milk has long ceased to be a matter for debate—it has become a religion. Among writers, especially lay writers, who feel that in a large way raw milk is not safe bacteriologically, boiled milk is barely mentioned and pasteurized milk, with its lower heating point, is given ream after ream. Incidentally, it is of interest that Jacobi weathered all these storms and always used boiled milk—and that in Germany and France boiled milk has been used practically exclusively for decades by pediatricians quite untouched by our discussions. The assumption that boiled milk only is used because of the poor milk supply in these countries hardly seems warranted. In countries like Germany and France where puericulture has become a national institution the production of pure milk for infant feeding purposes would be a small additional item if it did not already exist.

In the presence of such confusion of ideas it does not seem inappropriate to state the facts as we know them plainly and as far as possible without prejudice. The question of the relative position of raw, boiled, and pasteurized cow's milk in the problem of feeding the baby artificially can perhaps best be considered under the following heads as I have done elsewhere[21] and from which I quote freely:

1. Does raw milk offer advantages over boiled milk?
2. Does boiled milk offer advantages over raw milk?
3. Does pasteurization solve the problem?
4. Does certified milk solve the problem?

1. *Does Raw Milk Offer Advantages over Boiled Milk?*—The most obvious advantage of raw milk would seem to be its rawness. Mother's milk is raw—all milk is raw when taken under natural conditions. In giving the baby the milk of another species, say of the cow, we can, however, no longer assume that rawness is of value, or essential, or so essential that the advantages cannot be overweighed by other factors. While rawness may be desirable, as it certainly is for the young of each species when its own mother's milk is concerned, and while the absence of rawness may lead to some trifling deviation from normal progress, this very rawness may on the other hand bring some greater disadvantage such as the presence of harmful bacteria, or a large hard curd that is difficult to digest. The question cannot be settled by an appeal to nature, for we have already departed far from nature when we substitute cow's milk for breast milk—the appeal must ultimately be to the baby itself.

More specifically stated, raw milk is known to contain a large number of enzyms, together with antitoxins and antibodies and also certain vital principles, called vitamins, that are known to be necessary for normal growth and health. It is also known that some of these are destroyed or weakened by heat, especially by prolonged heat. Concerning the *enzyms* Heinemann[20] says: "It must be confessed that our knowledge of the origin of milk enzyms has not reached the point where it can be positively stated that certain enzyms originate from one or another. There is little doubt that some enzyms that have been found in milk are really of bacterial origin, but the existence of inherent milk enzyms still awaits positive proof. It has been assumed that some enzyms in milk are an aid in digestion and special stress has been laid upon this point in reference to the use of cow's milk for infant feeding. It is doubtful, however, whether enzyms in cow's milk are of real importance to the human infant, for it is natural to assume that if inherent digestive enzymes actually exist in cow's milk they would be of little value for another species of mammal." Clinically, it can be safely stated that we have never been aware of the presence, or noted the absence of these enzymes in feeding cow's milk to babies.

The question of *antibodies, immune bodies, antitoxins,* etc., in cow's milk can of course be of interest only to the calf, for the baby has a morbidity that has few if any points of similarity with that of the calf.

When we come to consider the relation of heat to vitamins, we must think of them in our present knowledge as in two classes: those that McCollum and his co-laborers have designated as *fat soluble A,* and *water soluble B,* that are necessary for health in young animals and for normal growth and for the prevention of certain morbid processes; and on the other hand the *anti-scorbutic vitamin C* which is likewise necessary to insure normal growth and health and to prevent scurvy.

With reference to the former I will quote from McCollum[4] (p. 136) as follows: "Osborne and Mendel have shown that butter fat may have a blast of steam passed through it for two hours and still retain its peculiar growth promoting properties due to the presence of the *fat soluble A*. . . . McCollum and Davis have pointed out that wheat germ can be moistened and heated in an antoclave at 15 lb. pressure for an hour or more without any extensive destruction of the water soluble B." They have even found the former active in condensed and evaporated milks, and the latter in canned goods. That boiling the milk does not affect the growth promoting fat soluble A of the milk, which alone directly interests us here, is again a matter of clinical experience.

Ultimately all discussion as to whether the heating of cow's milk is harmful to babies naturally comes back to *scurvy*, the only condition concerning which any real evidence has ever been brought forward that heating may be harmful (see "Scurvy" for details). It is chiefly in this country that scurvy insistently obtrudes itself for reasons that are obvious from what has been said in the preceding pages. It is also largely due to the fact that in 1898 a collective report[22] on scurvy was made to the American Pediatric Society by a committee of that body in which it was shown that the largest number of cases occurred when fed proprietary foods, a very few on cooked milk, even fewer on raw and on breast milk. In the final conclusions heating was not mentioned and a minority report by one member contained the clause, "Sterilizing, pasteurizing, or cooking of milk food is not *per se* responsible for the scurvy condition." In spite of this the heating of milk has ever since this report been advanced prominently as a factor in the production of scurvy. Largely as a result of this has sprung up the insistent demand for raw milk, in this country and in this country alone. In France and Germany raw milk is literally only of didactic interest, and yet all reports that come from these countries where boiled milk is used exclusively in feeding babies seem to indicate that they have less scurvy than we, or at least no more. French observers like Budin, Variot, Anderodias, Maygrier and others tell us that they have fed at their consultations for nurslings many thousands of infants for long periods of time on milk boiled, not five minutes, but 30 minutes or longer, without seeing a single case of scurvy. In 1910, 12 years after the report to the American Pediatric Society, Anderodias[25] said: "In the consultations for nurslings in which we feed sterilized milk as in those of Budin, Variot, and Maygrier, one has yet to see an infant with scurvy. . . ." The questions too would naturally arise why not at least *one* case of scurvy should have occurred—and also why the subsequent literature of France and of Germany does not reveal the presence of a larger number of cases. After all it is idle to compare the incidence of scurvy in France and Germany as compared with this country as a measure of the part that boiled and raw milk play. The important factor in the causation of scurvy in this country is neither boiled or pasteurized milk, nor raw milk, but certain proprietary foods that are used without fresh milk and these are not used to the same extent in Germany and France. It is, however, of significance that scurvy is not especially frequent in these countries in spite of the fact that they use boiled milk almost wholly.

I might quote[9] again in this connection a case of my own in an infant nine months old that had a typical scurvy with exquisite pain and tenderness of the lower part of the femur, and as florid a scorbutic gingivitis located *only about the teeth* as I have ever seen. On undiluted certified whole milk carefully boiled five minutes in a sauce pan, with no fruit juice or food of any other kind, and under unquestionable observation, the recovery was as rapid as any I have ever seen in which all the anti-scorbutic armamentarium was used. I might also state that the very last case of scurvy I have seen had never had other than raw milk.

The question of what constitutes and also what causes scurvy has in recent years been brought to the front by Hess[24] in a very original manner as a result of animal experimentation, and what is far more important, of observations of infants in an admirably conducted orphan asylum where numbers and conditions were most favorable for this study. While his conclusions would seem to require confirmation and further detail they are of great interest in this connection. Contrary to the usual interpretation of scurvy he includes as latent or subacute scurvy "anemia, pallor, failure to gain in weight, rapidity of pulse and respiration, tenderness of the bones and a sharp recession of all these symptoms when orange juice or other anti-scorbutic foodstuff is given." Hess[24] found that a certain number of children fed exclusively on pasteurized milk formulæ for five or six months developed scurvy in the above sense—more so if the milk was heated to 165°F. than if heated to 145°F. He found, however, that staleness of the milk, i.e. length of time after pasteurization, was of greater importance than the degree of heating. When the milk was obtained fresh and raw and then pasteurized at the institution and fed at once scurvy did not develop, and one case of "subacute" scurvy that had developed on stale pasteurized milk "improved" on the home pasteurized milk. By keeping the pasteurized milk for 48 hours he could again produce scurvy. This was also the case with raw certified milk kept on ice for 48 hours before feeding. According to Hess also "it would seem that milk which has been boiled is less apt to induce scurvy than milk which has been pasteurized." He found also that dried milk lost little of its anti-scorbutic properties and that canned tomato juice and orange juice boiled 10 minutes were still strongly anti-scorbutic. In the presence of all this newer evidence on the question of the relation of heat to the anti-scorbutic factor in milk one is tempted to throw up one's hands and approach a more positive phase of the subject, for after all what difference does it make practically?

The one fact about scurvy, whether latent, subacute, or florid, before which all doubt and all theory are laid aside is the fact that it is the most easily and quickly curable and the most easily preventable disease that we know. Orange juice will convert a tortured and apparently desperately sick baby into a comfortable and well baby within a few days and the baby that gets a few teaspoonfuls of orange juice each day after the third or fourth month of life will never develop any form of scurvy. To one who is investigating the pathogenesis of scurvy the relative incidence of scurvy on raw, fresh, stale, boiled or pasteurized milk is of great interest. In the practical feeding of babies it is of no interest whatever for no one has ever contended that a small amount of orange juice is not a wholly adequate antiscorbutic. If there should seem, then, to be some good reason why milk should be boiled before it is fed to a baby surely the hobgoblin of scurvy will not scare us from it.

It is also known that boiling produces a series of changes in milk such as caramelization of some of the milk sugar, coagulation of the lactalbumin, changes in the salts, and other changes that might impair its nutritive value as an infant food, or might lead to some form of disnourishment that would not occur if the milk were given raw. The development of rickets would first suggest itself as a possibility. Hardly a single textbook, British, French, German, or American, however, even mentions rickets in connection with boiled milk. As to other conditions one can only say that there is no known nutritive disorder that comes from boiling the milk if one may dismiss scurvy from consideration. Boiling the milk of its own species can of course be assumed to be harmful to the young of that species, but cow's milk is for the baby an unnatural, non-specific food, and boiling can easily lead to benefits that overweigh the disadvantages, if they exist. Again the appeal must be to the baby, not to theory. Fortunately, we have the baby's answer from an unbiased source. Janet S. Lane-Claypon[26] was appointed some seven or eight years ago by the Local Government Board of London to investigate "the Available Data in Regard to the Value of Boiled Milk as a Food for Infants and Young Animals." In her very exhaustive report that included all available experimental and clinical data on the subject, and a personal investigation of the results both in England and in Germany, she sums up the question in the final "Summary" by saying that "such small differences as have been found in the nutritive values of raw and of boiled milk have been in favor of boiled milk." While the subject can hardly be settled by such data they nevertheless furnish the most tangible evidence that can be presented that so far as we know at the present time boiling does not seem to impair the nutritive value of cow's milk as an infant food.

Among other advantages assumed for raw milk is that it has certain germicidal properties, that not only, for a time, at least, inhibit the growth of all bacteria, but that they ultimately determine a bacterial growth that is non-pathogenic; that boiling the milk destroys these properties, and also destroys the normally developing lactic acid producing bacteria that determine the natural bacterial growth in milk, and that milk so altered decomposes instead of souring normally. These products of decomposition are assumed to be pathogenic. As to the germicidal properties of raw milk I will quote again from Heineman[20] (p. 256): "Whatever the true cause or causes of the so-called germicidal properties of fresh milk may be it seems certain that it is too feeble to be of practical value. It cannot be used successfully as an argument against the practice of heating milk for the purpose of destroying pathogenic bacteria, even though antibodies are partly destroyed at the temperature required for pasteurization. As a matter of fact the germicidal action disappears as a rule after a lapse of 12 hours, so the keeping quality of milk is not materially prolonged." As to the decomposition of boiled milk, and the same thing would apply to pasteurized milk, it would seem that this has been of considerable theoretical, but of very little practical, interest. None of us seem ever to have run across it, or at least to have recognized it. Boiling of milk furthermore does not carry with it the assumption that all the usual safeguards to prevent milk from spoiling shall be abrogated. Moreover, it is evident that the only logical course that commends itself, both in this connection and in its possible relation to the pathogenesis of scurvy, is not to boil the milk till it reaches the ultimate consumer, if it is to be boiled at all. Identically the same reason applies to pasteurization.

A final and almost universally voiced objection to boiled milk is that it is constipating. In a matter of this sort as we well know it is most difficult to separate tradition from experience. Breast milk is laxative—cow's milk *per se* to the baby is constipating unless it leads to indigestion and diarrhea. A formed, alkaline stool is as natural to the thriving cow's milk fed baby, whether the milk is raw or is boiled, as is a soft, mushy, acid stool to a breast fed baby. The baby that is fed on cow's milk, raw or boiled, and is reasonably constipated is digesting its milk perfectly. The only exception to this is the fat constipation of Czerny and Keller (Milchnährschaden). This condition can be *avoided* with boiled milk as easily as, if not indeed more easily than, with raw milk, because a larger amount of carbohydrate can be added to boiled milk than to raw milk without danger of indigestion.

That boiled milk is somewhat more constipating than raw milk seems probable, that it is so to an annoying extent in proper therapeutic amounts I have never been able to satisfy myself. It is hard to tell where the baby leaves off and the mother begins in the proper evaluation of this symptom. In digestive disturbances, with a tendency to diarrhea, boiled milk is certainly more constipating as we have always known. Personally, I think it is far more correct to say that under these circumstances raw milk is more laxative, because more indigestible, than that boiled milk is more constipating, because more easily digested. I might quote from a source that is certainly not prejudiced in favor of boiled milk. Morse and Talbot say (p. 309):[23] "The heating of milk unquestionably predisposes to constipation, but only to a slight extent. It is a far less common cause of constipation than is ordinarily supposed." Dunn[26] says (p. 498): "The heating of milk especially if the milk be boiled or sterilized instead of pasteurized, may be a cause of constipation, but this cause is less common than is generally supposed, and has never actually been proven." Even if one may grant that boiled milk is relatively more constipating than raw milk, this is so minor an evil as compared with even moderately loose stools which, in the cow's milk fed baby, are incompatible with normal progress; and the condition is so readily remedied by the various measures with which we are familiar, and so naturally corrects itself as the older infant gets a more varied, less milk, diet; that it does not deserve the serious consideration it has received if there are advantages in boiling that more than compensate for this slight difference.

2. *Does Boiled Milk Offer Advantages over Raw Milk?—Two advantages,* and only two, suggest themselves. The first needs little exposition, it is self evident. *Boiling destroys practically all pathogenic organisms and up to the present time it is the only reliable measure that does do so.* The array of diseases that have been marshalled forth as capable of being milk borne, the many epidemics of typhoid, dysentery, scarlet fever, streptococcus sore throat, and enteritis, and others that have been traced to milk, are certainly a serious factor in this problem. So long again as it has not yet been disproven, to take a single instance, that a large part of all human tuberculosis is taken in through the alimentary tract in infancy so long it is manifestly necessary to use that theory as a working hypothesis in this connection. The work of Evans,[28] and of Fleischner,[29] and others on the Bacterium Abortus is suggestive of other possibilities that are still not measurable. These investigators found the Bacterium Abortus which causes abortion in cows in nearly all raw cow's milk that they examined in this country. They have, moreover, shown that this organism is morphologically, and in animal experimentation as shown in pathogenesis and in agglutination and in specific cutaneous hypersensitiveness tests practically identical with certain strains of Bacterium Melitensis, the cause of Malta fever in human beings and of abortion in goats, in the milk of the latter of which the *Bacterium Melitensis* was found in 10 per cent. of cases by the British Commission that investigated the matter. While evidence of human pathogenicity of the Bacterium Abortus is wanting, Evans pertinently asks: "Are we sure that cases of glandular disease, or cases of abortion, or possibly diseases of the respiratory tract, may not sometimes occur among human subjects in this country as a result of drinking raw cow's milk."

The second advantage of boiled milk is purely physiologic. We have long known, clinically and empirically, that when a baby has a diarrhea or an indigestion from whatever source, that boiled milk, if milk is to be used at all, works better than raw milk. The answer to this is glibly given—"boiled milk is constipating." When we ask "why it is more constipating than raw milk" the only available answer that we have is, "Because it is more easily and quickly digested, liquefied and absorbed than is raw milk." If it is more easily digested in the presence of a digestive disturbance it must also be more easily digested by a normal baby. If it is more easily digested than raw milk, and there are no counterbalancing objections, then why not avail oneself of that aid in the difficult problem of adjusting cow's milk to the baby?

Clinical observers, who have fed raw milk for years, and have fed boiled milk for an equal number of years, are quite agreed that boiled cow's milk is easier to digest than raw milk. The problem of feeding a baby becomes measurably simpler with boiled milk, and one can always proceed more quickly to larger amounts of greater concentration than with raw milk. This clinical observation, which in my own case preceded all experimental observations, is readily accounted for by the latter. To one who has seen as I have, over and over again with mathematical precision, the hard, tough, rubbery curds as large as a walnut, or even larger, that have been returned from the stomach after the ingestion of raw cow's milk, and with raw milk always and alone; and then with equal regularity, the fine, soft, porous, friable curds the size of a pea or less that are returned with boiled milk; no other explanation would seem necessary to account for this clinical difference. Whether the boiling has some other effect on cow's milk that makes it more easily digestible by the baby is not known. Personally I am inclined to think it has, possibly by some action on the fat, possibly in some other manner. Certainly the baby that is fed boiled milk can be given more cow's milk fat than when it is fed on raw milk, though this may have to do wholly with the greater digestibility, and hence lessened tendency to indigestion, of the boiled milk casein. The addition of sodium citrate to the milk, too, prevents coagulation, but it has always seemed to me, that it had a less decided effect than boiling especially in babies with a tendency to diarrhea.

Two peculiarities of *all* cow's milk stomach curds have already been mentioned but require emphasis here.[7] For two hours they increase in size, and all digestion is purely peripheral, the curds never disintegrate and get softer, but always and continuously harder. The real criterion then is the condition at the end of two hours, when they have reached their maximum size and begin to get smaller. I think the ratio in size alone, of the curd of boiled milk to that of raw milk, at this maximum two-hour period, can conservatively be placed at 1 to 30 or 40. A nearer criterion, however, is not the size of the curds but the relative lengths of the radii from the surface to the center of the curd, that represent the distance that digestion must travel. This ratio could be placed at perhaps 1 to 5 in the larger curds. This however does not represent the true digestive task, for with raw milk there are commonly a few very large curds and only a small amount of smaller curds, while with boiled milk there is a large amount of fine detritus and only a few relatively larger curds. If this factor is included a ratio of 1 to 10 would probably more nearly express the facts. To this must be added the further fact that the raw milk curd is much harder than the boiled milk curd even after two hours, though conclusive data as to the relative digestibility of the two kinds of curd remain to be ascertained.

Since these considerations do not wholly remove the problem from the domain of theory, the appeal must again be made to the baby, that knows no theory. From the baby itself we have two answers.

In the first place almost all therapeutic measures that have been employed to make cow's milk more acceptable to the baby have one thing and only one thing in common, intentionally or unintentionally they make for a smaller and commonly softer and more porous curd. A mere summarization of these measures is not without interest: Dilution with water; the use of cereal waters; of alkalies and salts such as lime-water, milk of magnesia, sodium and potassium bicarbonate and sodium citrate; the use of boiled, condensed, evaporated, and dried milk, including such baby foods as Nestle's, Malted Milk, and Ramogen; the precoagulation of milk with rennin as in the use of pegnin and chymogen, with lactic acid bacilli as in the use of buttermilk, or acidified milk, and the combination of both in Eiweissmilch; the use of malt soup; of whey reduced and whey adapted milk, and all the other attempts such as Beidert's and Meig's and Rotch's to make a "one formula milk" similar to mother's milk; the use of high cream and low protein mixtures, and that of whey and the split protein; the addition of dried casein in the forms of larosan, sanatogen, and plasmon. This list, which nearly exhausts our therapeutic armamentarium, would lead one to think that the baby has let us know unmistakably that he prefers his curd small and if possible soft, as in his mother's milk, *and that he is not particular how that end is attained.*

The second answer is the hard "casein" curd of the stools. When a baby gets raw milk in excess of its casein tolerance it quite frequently happens that the hard curds that are formed in the stomach pass undigested clear through the whole digestive tract and appear in the stools as the white or amber colored hard stools.[19] With this appearance of hard curds there is nearly always a coincidental indigestion with looseness of the bowels of varying degree. Whether this is due to the casein, as seems probable, or to some other factor that causes raw milk indigestion does not really concern us here. The important fact is that if the milk is now boiled the curds disappear and with them the indigestion. That such curds are apparently a disturber in digestion, possibly purely mechanical in nature, not unlike the action of corn, peanuts, bran, etc., is evidenced by the well known fact that *Eiweissmilch* fails to give its favorable action if the fine casein coagulum that forms a part of its composition is too coarse. The fact that hard curds occasionally, but very rarely, occur in a normal or nearly normal stool is easily explained by the fact that not infrequently one or more larger curds are formed in the stomach by an accidental combination and that these pass through without causing trouble as would a penny or a safety pin. It is perfectly evident on the other hand that a child may have a troublesome casein indigestion, vomiting, discomfort, etc. and yet digest all of the casein masses before they reach the stool, i.e., the appearance of curds in the stools represents rather a severe condition.

It must be stated that a fairly large percentage of babies will apparently digest raw milk as well as boiled milk within therapeutic limits. This is not synonomous with saying that raw milk casein is as easy to digest as boiled milk casein. Many normal babies have a tolerance far in excess both of their natural food and of the usual modifications of cow's milk that we prescribe for them. It must be borne in mind too that when we speak of a baby's tolerance for raw milk we commonly mean for a raw milk formula in which the casein has been modified in various ways, nearly always by dilution, and commonly by the addition of such other casein modifiers as cereal waters, lime-water, etc. *Boiling is simply an excellent casein modifier and it is the only completely effective solution of the bacteriological problem.*

3. *Does Pasteurization Solve the Problem?*—Pasteurization was devised so that milk could be freed from pathogenic organisms at a lower temperature than that of boiling, with the idea that by so doing the harmful effects of boiling might be avoided. It is a complicated procedure that requires accurate technique. It has practically been discarded from the home, complicated devices being unfeasible and the many simple procedures wholly unreliable, and this especially in the homes where it would be most needed. Commercial pasteurization is as yet not wholly reliable and for some of the very reasons for which it was devised it defeats its purpose. Such milk has to be kept 24 to 48 hours until finally consumed. We have seen that this is an item, possibly an important one, in the causation of scurvy.[24] Such milk too is just as liable to decomposition after pasteurization as after boiling, which has been advanced as an argument against boiling. Boiling has here the great advantage that it is done at home just before the milk is consumed, while in the case of commercially pasteurized milk there is an unavoidably much longer lapse of time between pasteurization and consumption. This period too withdraws itself from careful scrutiny and during this time the milk is in very unbacteriological hands and commonly not continuously on ice. This is probably a minimal danger but it is a danger that exists with pasteurized milk and not with home boiled milk if it exists at all.

The relative activity of the anti-scorbutic, and other allied properties, in pasteurized and in boiled milk which is after all probably the activating idea back of pasteurization remains to be more sharply defined. At present it is based more on tradition and assumption than on evidence, and demonstration is wholly wanting. If the evidence that Hess[24] has furnished can be accepted then boiled milk has a double advantage in that it is said to have been found more antiscorbutic per se than pasteurized milk, and it becomes very much more so if the factor of staleness is an item as it has always seemed to be. Pasteurization implies, and probably always will, pasteurization at a distant central plant; boiling implies not a distant plant, but a sauce pan and that in the home the day the milk is consumed.

At the present time mothers often object to boiling the milk "because of the trouble it takes." This is partly because it has not become a recognized routine of the household; and partly because tradition is tugging at their elbows at every turn in the form of the baby book, the Government book, and the lay writer.

Finally, even if pasteurization were of vital superiority to boiling from a biological standpoint, as it is admittedly not from a bacteriological standpoint, the physiological problem would remain untouched. Physiologically, i.e., in coagulation behavior, pasteurized milk is essentially like raw milk.[7,9] Clinically the pediatrician, if for the moment we may set scurvy aside as undetermined, can point to no single difference between boiled and pasteurized milk as it affects the baby's nutrition, that would make him willing to break a lance in defense of pasteurization. Even if scurvy still intrudes itself we always have orange juice, or tomato juice, at hand.

4. *Does Certified Milk Solve the Problem?*—There can be but one opinion on the question of whether a food for infants that is an excellent culture medium, that is handled commercially by men not bacteriologists, that is furthermore peculiarly subject to bacterial contamination all along its devious way from before it leaves the cow till it enters the baby's mouth, should be safeguarded by all the means advocated by our milk commissions. One of the great benefits that have come from the memorable step that lead Coit in 1892 to look for a safe milk for his own baby and ultimately a safer milk for all babies lies in the fact that this agitation has resulted in a betterment of all milk. Constantly better and safer milk can be our only ideal. At the present time certified milk is accessible to only a small minority of babies except in larger centers of population. Even here it is of necessity so expensive that it does not reach extensively that stratum where it would seem to be most indicated. That ordinary milk under present conditions of production in a large city can be made a safe food for infants if it is boiled in the home has been amply demonstrated by the Infant Welfare Society of Chicago in that very stratum of the social scale. For years only commercial home boiled milk has been used with results that seem little, if at all, inferior to those obtained under the most favorable circumstances. That, in recent years, the milk has first been commercially pasteurized can only add weight to the present consideration. The vast majority of babies do not and cannot receive certified milk and if boiling seems indicated in this majority and does no visible harm, then it would seem equally harmless in that minority that uses certified milk if it should seem indicated for other reasons.

The object of certified milk is to furnish a clean, fresh milk, and especially one that from a bacteriological standpoint can safely be used raw in feeding babies. It assumes a reliable and ever vigilant milk commission and honest and efficient producers and carriers. Any weak point in this human chain can leave us in a fool's paradise. This danger may be small but it is ever present. Bacteriologically, alone, certification does not even remotely approximate boiling. Biologically we have seen there is still much confusion in our views and findings. Physiologically certified milk is *raw milk* with a curd so large and hard and peculiar that it can safely be classified as the most uniquely solid food that we ever give a young infant. *For this reason it is just as reasonable to cook clean, fresh, certified milk as it is to cook a clean, fresh potato, or a turnip.* In both cases a very solid food is made less solid and more digestible.

Summary.—The whole question of whether milk should be used raw, boiled, or pasteurized in infant feeding can be summed up practically as follows: If one feels for natural reasons, that raw milk ought to be used in a given case, or as a routine, then one ought to be reasonably sure of the milk. If one is not sure of the milk it can be made safer from a bacteriological standpoint if it is boiled or pasteurized. Of these two procedures boiling is preferable both bacteriologically and biologically. It is more certain from the former standpoint, and possibly safer from the latter because it can much more easily and more effectively be done at home thereby lessening the factor of staleness after heating and possibly also other factors not yet clearly determined. If one uses boiled or pasteurized milk, or for that matter raw milk, it is well to begin small amounts of orange juice in the third or fourth month, or if desirable earlier, and gradually to increase the amount. Bacteriologically boiling in the home is the only *wholly* safe method. Biologically from all present evidence raw milk is somewhat more active, boiled and pasteurized milk so nearly alike that the difference is not clearly evident, and whatever slight differences there are among the three seem wholly equalized by the use of orange juice. Physiologically boiled milk is very different from the other two, and if one wishes to avail oneself of that difference in feeding babies there is no valid practical reason why one should not do so.

THE PRACTICAL ARTIFICIAL FEEDING OF BABIES

The student who enters for the first time the domain of practical artificial infant feeding, and the practitioner who is searching for further light on his feeding problems, is apt to be confused in the presence of what seems an endless array of differences of opinion and of practice, instead of finding that substantial agreement that characterizes many other branches of medicine. As a matter of fact the differences are far less real than they would seem at first glance, and are very frequently differences of method rather than of substance. Two men will feed with equal success the same baby by two methods that seem wholly different, and yet their fundamental ideas are practically in accord, just as two men will walk to the same place by two different routes. Discussions are sometimes biased, and giants often fade into windmills as they are approached more closely. The opinions of individuals and of schools commonly pass not for what they are but for what they were, or for what they are reported to be by others who have not fully informed themselves. A new discovery, or the exposition of a certain idea, that may mean relatively little to the author, is often distorted by scientific gossip into a form quite unrecognizable to the originator. In the exposition of a subject so complex, and with so many angles, the points of difference can easily be brought into prominence, rather than the points of agreement. There is no question that in the evolution of this subject there was a long period of increasing complexity. We have long since entered a period of increasing simplicity and consequently of agreement. I hope that this trend will be evident in the following pages.

In the exposition of our ideas of artificial infant feeding only *four ideas, or methods, or viewpoints,* stand out at the present time that require discussion. From time to time, first, the view still crops out, though increasingly less frequently, that babies can be fed successfully on *undiluted cow's milk.* Secondly, attempts have been made, sporadically, for 50 years down to the present time to devise some *one modification of cow's milk* that will so closely approach mother's milk that it can be given to all babies. The third viewpoint is that of the so-called *Percentage, or American, method* of feeding that at one time was generally accepted in this country as "a system of feeding" but has become so modified that it now differs from other methods of feeding essentially only in that the representation of each food element in a milk mixture is visualized and expressed in terms of percentages. The fourth method, which would seem more nearly to represent the present status or tendency of infant feeding, will be developed in detail as the basis of our discussion of the practical feeding of babies. It consists primarily, *in the simple dilution of milk with the addition of carbohydrates.* It differs from the percentage viewpoint in its greater simplicity, but perhaps finally only in that it deals rather with *amounts* of milk, sugar, etc. than with *percentages* of the different food elements.

The Feeding of Undiluted Whole Milk.—Except in France and to some extent in England this return to the primal method of substituting the unmodified milk of the cow or the goat for the natural food of the baby has had little vogue. In this country the method is practically untried and unknown, except by certain followers of the English method. In Germany sporadic attempts have been made from time to time only to fall into disfavor. Even in France and in England it is not correct to say that unmodified milk was used. In France especial emphasis was always laid on "lait sterilizè," the milk being cooked for a long time. In England the practice was always accompanied by the use of 1 or 2, commonly 2 gr. of sodium citrate to each ounce of milk. Usually too the undiluted milk was not given to younger infants.

Thus Budin[30] who was the most ardent and most quoted feeder of undiluted milk in France recommended one-tenth of the baby's weight daily of sterilized milk and reports excellent results in "infants after the fifth or sixth month and weighing 13 to 15 lb." While undiluted milk is wholly unfitted to the young infant, as is generally agreed, both because it is hard to digest and also because of its undesirably high protein content, it can probably be given in this form much earlier than is generally thought if it is boiled, or alkalinized, or precoagulated by rennin, or by lactic acid forming bacilli. It has latterally become my own custom to put many of my babies on undiluted boiled milk by the eighth or ninth month though I see no special advantage in the procedure except in the baby that wants only four bottles a day.

Percentage Feeding.—The second step in the modification of cow's milk was to dilute it with water to make it act more like human milk. With advancing knowledge of the comparative chemistry of human and of cow's milk and with the natural view that like composition should lead to like results, for many years the aim of advanced pediatricians was to produce a modification of cow's milk that would be sufficiently nearly like human milk to make it nearly as acceptable to the baby. The pioneer in this movement was Biedert.[1] Some 40 years ago he developed his Rhamgemenge (cream-mixture) in which the protein, which he considered the essential point of difference between human and cow's milk, was reduced to about 1 per cent., approximately that of human milk, by diluting 10 per cent. cream with three parts water. The percentage of cream so obtained was about 2.50 per cent., and the percentage of sugar was increased to 5 per cent. by the addition of milk sugar. To this basic mixture whole milk was to be added after the first few months in increasing amounts according to a table of six steps to be used as a variable guide for different months and for different babies, until the protein was raised to that of whole undiluted milk in the early part of the second half year of life. This mixture was also prepared commercially, for purposes of uniformity of composition, in the form of a condensed product which under the name of Ramogen is still in use.

Meigs, of Philadelphia, having found empirically in an extensive practice with babies that certain proportions of milk, cream, lime-water, water, and milk sugar seemed to agree better with the average baby than any other artificial food, had this mixture carefully examined chemically. He found that in the one important food element that was held to distinguish cow's milk from human milk, the casein, the percentage was very nearly the same in this mixture as in human milk, and that the percentages of fat and of milk sugar were approximately the same as in the latter. He further perfected this mixture until it was as nearly like human milk chemically as the knowledge of chemistry at that time would permit. This mixture, commonly, known as "Meigs' Mixture," was made by mixing 1 oz. of milk, 2 oz. of 14 to 16 per cent. cream, 2 oz. of lime-water, and 3 oz. of a milksugar solution (17¾ dr. of milksugar to one pint of water) and had about the following percentage composition: Fat 3.50 per cent.; protein 1.21 per cent.; sugar 6.66 per cent.; ash 0.25 per cent.

The work of Meigs was carried on and developed by Thomas Morgan Rotch to whom is due the elaboration and popularization of Percentage Feeding. Rotch at first merely aimed to make the composition of Meigs' Mixture more nearly like that of breast milk, by using only one-fourth as much lime-water. His formula was: Cream (20 per cent.) 1½ oz.; milk 1 oz.; water 5 oz.; milksugar 3⅜ dr.; lime-water ½ oz. When this formula was found not to fit all babies Rotch took the further step that became and remains the underlying principle of Percentage Feeding. Just 30 years ago (1889) Rotch in the concluding paragraph of his chapter on "Infant Feeding in Keating's "Cyclopedia of the Diseases of Children" (p. 329) took this step when he said: "See that the analysis of the food corresponds as closely as possible to that of human milk. . . . If this is not successful refer to the control record and adapt the food to any material idiosyncrasy shown by the record. If no control record has been kept experimentally try to discover the especial idiosyncrasy of the individual infant by changing the percentage of the fat, sugar, albuminoids, or ash."

The succeeding steps in the evolution of Percentage Feeding, as elaborated by Rotch and his followers, naturally followed. The single formula unlike mother's milk did not fit all babies even if of the same age and perfectly healthy; healthy babies of different ages seemed to require or tolerate different percentages of the food elements; and the baby with a digestive disturbance required a wholly individual adjustment quite unlike that of the healthy baby. To meet these points the healthy baby was tested out by a weak formula and all the elements were gradually increased to what seemed the desirable amount. This desired amount was shown by the progress of the baby, and also by its conformity with certain elaborate tables based on experience, that were drawn up to indicate the proper percentages and amounts usually tolerated by healthy babies of given ages, to be used as variable guides by the beginner. For the baby with a digestive disturbance it was further necessary to learn what symptoms characterized the overstepping of tolerance for the different food elements, so that the food could be adjusted with reference to that particular element if such symptoms arose. It further became necessary to devise methods for calculating what amounts of the different ingredients of known composition, such as 16 per cent. cream, 4 per cent. milk, fat free milk, whey, sugar, etc. were required to give the desired percentages of each food element, and also, obversely, to be able to reduce to percentages any given formula. This difficult procedure led to simpler calculating devices some of which, though algebraic in form, were marvels of efficiency and simplicity. Unfortunately, the calculations necessary to arrive at, or reduce to percentages, the food formula still seemed very complicated and "simpler methods" appeared in constant succession, methods that simplified the matter only to the originator, and left the practitioner bewildered in the presence of what seemed like mathematics rather than practical dietetics.

The natural corollary to all this mathematical complexity was the establishment of the Walker-Gordon Laboratories, first in Boston and later in many other larger cities of the East and Middle West. To these laboratories it was only necessary to send prescriptions for food containing given percentages of the food elements, together with the number of bottles desired and the amount of food in each.

In the early development of Percentage Feeding the casein always held the center of interest. The curds found in the stools were always considered an indication of overstepping of casein tolerance, and naturally called for greater reduction of the amount of casein. When the protein reached a point so low that further reduction would leave less than the amount demanded by nature as shown in human milk, and also as a preventive of casein indigestion in general, the whey proteins were often increased by the addition of whey as part of the diluent. The resulting protein, commonly called the "split-protein," resulted in a greater representation of the soluble proteins in the mixture as compared with the casein than would be given in simple dilution of milk or cream. The fat and the sugar were long considered relatively unimportant in the causation of digestive disturbances, and the attempt was always made to increase them as rapidly as possible to the amounts found in breast milk. Cream always naturally entered into the formulæ, because the fat was given in larger amount than the protein. The establishment of the fact that the common curds are fat curds and not casein curds, later led to a great modification of these views.

In more recent years the casein is no longer the *bête noir;* the protein in rarely split as a result; the fat and the sugar have been given their proper place in the pathology of artificial feeding; the importance of measuring the food in terms of amounts, or of calories, and not alone in percentages is recognized; the chronological tables have nearly disappeared; and the Walker-Gordon Laboratories have died a natural death except in the far East. Emphasis is now laid on the fact that Percentage Feeding is not a system, or method of feeding, but "simply a method of recording the characteristics of the food given to the infant . . . a system of record that gives a common terminology in infant feeding, and a common basis by which various methods of modifying milk may be compared both as to theory and as to practical results."[27] Upon this basis alone can it be examined critically.

The first obvious reflection would seem to be that while the percentage of a given food element is of value from a purely digestive standpoint, the total amount of food taken in 24 hours is the really important thing from every point of view. Even the breast fed baby with a milk of constant percentages of food elements that is thriving normally on 20 oz. of milk a day can be seriously upset on 30 oz. With artificial food the harmful response is immeasurably more prompt and certain. To add to the calculations necessary to determine the percentages of food elements those necessary to determine the amounts, or caloric value, of so complex a mixture would seem to add the last straw.

A second consideration would seem to lie in the fact that there is nothing in infantile digestion or metabolism that corresponds either in kind or in amount to so minute and accurate a fractional dosage of the different food elements, and especially the protein, that elaborate calculations are necessary to adapt them. Moreover, the variations in the composition of the food stuffs used, and in the measurements made in the practical preparation of the food mixtures, except perhaps in laboratories, would largely nullify all finer calculations.

Again, with the exception of the fat the various food elements can be varied quite as effectively and as accurately in the use of simple milk dilutions with the addition of carbohydrates as in percentage mixtures, at least within the limitations placed by the needs of the baby. The protein, with the casein as readily modified as it is, and with almost no evidence that it is a factor in metabolic disturbances, becomes a nearly negligible factor in both digestion and metabolism, provided always that there is enough. This is amply provided for in all simple milk mixtures. The carbohydrates are varied in one as in the other, except that in one case one thinks of amounts, in the other of percentages, though there is no obstacle to thinking in percentages in either case. The fat alone cannot be increased or diminished, except *pari passu* with the protein, and cannot approach the normal 4 per cent. of human milk unless nearly straight milk is used. This objection is practically removed when we consider that with percentage mixtures the tendency is to keep the fat considerably lower during the first few months than that of undiluted cow's milk, and that the latter is not approached until a time when only slightly diluted cow's milk can safely be given in which the percentage of fat is not much lower than it is in cow's milk or in percentage mixtures. When we add to this the generally accepted fact that the fat is the really difficult thing to digest in cow's milk, that it decidedly delays the emptying of the stomach and that it leads to serious digestive and metabolic disturbances, this objection is of little moment. This is all the more true when we bear in mind the fact that the closely allied and more easily digested carbohydrates can replace to an adequate extent the slight deficiency in fat. If there should seem to arise reasons why more fat should be given than is represented in simple milk dilutions, or if in a given case a larger amount is desired, this can easily be obtained by the addition of a little cream, or the use of top milk, and that without finer modification than the baby would seem to indicate. After all it is a question of baby and not of percentages. How closely the two methods approach is evident from what has been said. The percentage feeder may even say, as has been done many times, that the chief difference lies in the fact that one method is accurate, the other not. This point is not well taken. The baby responds to amounts of food, not to percentages, except in a restricted sense. The real difference lies in the view point and in the degree of simplicity. This simplicity, moreover, does not carry with it any inaccuracy that is reflected in the baby.

This brings us to a final serious consideration. The subject of infant feeding is not in great favor with the practitioner who feeds the great majority of babies and the fault lies in a large measure with the exponents of percentage feeding. Rightly or wrongly he gets the impression that the subject is of such complexity that it requires extensive mathematical calculations to apply it in practice. The ever new additions to this volume of mathematics have not reassured him. Ultimately he chucks it all for the simple patent food formulæ, or uses simple milk dilutions with some form of carbohydrate that is for the time most advertised. The consideration that an intelligent physician can master the technique in a short time, as he can that of surgery or obstetrics, is beside the question, the practical fact remains that he does not do it and never will. As a result percentage feeding has followed a course that is typical of all advances in infant feeding. At first rightly hailed as a great step in advance in scientific, or what is even more important, practical infant feeding, it was soon accepted by nearly all American Pediatricians, and recognized by nearly all practitioners as *the* method, whether followed or not, in the last decade to be steadily replaced by simpler methods until today it has only a rather restricted geographical following.

Single Formula Mixtures. Whey Reduced and Whey Adapted Mixtures. S.M.A. (Gerstenberger).—We have seen how Biedert, Meigs, and Rotch occupied themselves with perfecting a *single formula mixture* that would fit all babies, as does mother's milk. Their attention, as also that of later investigators, was devoted almost wholly to the protein, the fat, and the sugar, leaving the salts to only such modification as the adjustment of the other food elements carried with it. It became evident that these efforts did not solve the problem, and the salts became the center of interest in the single formula mixture.

Whey Reduced Milk (Friedenthal).—Friedenthal some years ago first attempted to modify not only the fat, protein, and sugar, but also the salts so that they would approach those in human milk, and thus help to make a mixture that would approach the latter in its behavior as an infant food. On the assumption, based on older and now known to have been in part incorrect analyses of human milk, that the sodium, calcium, and potassium salts were those chiefly in need of adjustment, Friedenthal constructed a food mixture to which he gave the name of Molkenreduzierte Milch (whey reduced milk). The whey salts were reduced in amount by the reduction of the total amount of milk. This reduced the sodium and calcium salts to the desired level, and at the same time the protein. The fat was raised by the addition of cream, and the sugar by adding milksugar. The relatively higher content of potassium salts in human milk was met by the addition of potassium salts. This single formula was made up of 330 c.c. of fat free milk, 600 c.c. of water, 58 gm. of milksugar, enough cream to make 4.5 per cent. of fat, and 1.8 gm. of a mixture of salts (Molkereizusatz) consisting of two parts of potassium chloride, one part of dipotassium phosphate, and one part of monopotassium phosphate.

Whey Adapted Milk (Schloss).[31]—Schloss later supplemented the work of Friedenthal by efforts looking toward a nearer approach to the salt content of human milk as shown in later analyses. He reduced the whey salts still further by using even less milk and cream than did Friedenthal; added however dried casein (nutrose, or Plasmon) to make up for the correspondingly decreased protein; added a dextrin-maltose preparation (Nährzucker) instead of milksugar to conform to newer ideas of carbohydrate availability; and concerned himself with the salts only as they were reduced by the greater milk dilution and with the rectification of the known Potassium deficiency. This preparation which he called Molkenadaptierte (whey adapted milk) had the following composition for each liter:

Milk	1/7 liter
Cream (20 per cent.)	1/7 liter
Nährzucker (without salt)	35 gm.
Nutrose or Plasmon	0.5 gm.
Potassium chlorid	0.2 gm.
Water	5/7 liter

This formula No. 1 was intended for younger babies. A formula No. 2 intended for older babies varied from this only in the use of 50 to 70 gm. of Nährzucker in place of the 35 gm. of salt free Nährzucker.

The formula of Schloss is perhaps not as well adapted as that of Friedenthal to determine the question of the value of whey adaptation itself. The addition of dried casein with its known fermentation-checking action; as well as that of a foreign sugar that ferments less readily than does milksugar, which is already more reduced by greater dilution of the milk and cream, introduce factors that are outside of the whey itself. That it has practical value in the feeding of healthy babies has been attested by many observers.

Synthetic Milk Adapted (Gerstenberger).[32]—Still more recently Gerstenberger and Ruh (1915 and 1919) have made a new attempt at a single formula mixture, which they have called S.M.A. or Synthetic Milk Adapted. Using the formula of Friedenthal as a basis they have modified it chiefly by the substitution of other fats for the fat of cow's milk to avoid the difficulties that are traceable to the latter in infant feeding. The fat of cow's milk is practically eliminated by using only fat free milk. The substitute fats consist of:

	PER CENT.
Tallow oil	10
Cocoanut oil	about 15
Cocoa butter	about 20
Cod-liver oil	10
Tallow	55 to 45

These fats are chosen to meet three requisites, outside of their digestibility: "(1) Saponification, iodin, Reichert-Meissl and melting point values to be like those of breast milk fat; (2) the presence of an adequate amount of the fat soluble growth factor; and (3) the use of cod-liver oil as a prophylactic against rickets. The whole mixture is homogenized ('Homogenized Milk')." With this single formula milk Gerstenberger and Ruh have reported "an excellent nutritional result, according to our present standards" in some 311 babies. Further observations by others must be awaited before S.M.A. can find its proper place in our feeding armamentarium. This preparation is now on the market in dried form.

The search for a *single formula food* for babies is certainly alluring and perhaps not wholly fatuous. It seems at least to define more sharply the requirements and limitations of artificial feeding. So far it occupies a place not unlike the quest for the creation of life from the non-living that has long occupied some of our more venturesome biologists. While important results are often heralded, ultimately by no stretch of the imagination has life ever been created without previous life, nor has human milk ever been created from cow's milk. The newer ideas that have been added to our knowledge of nutrition during the last decade however do not leave us wholly without hope that the "biological" factor that now seems to separate cow's milk from human milk may after all be chemical, and so capable of solution.

The Use of Simple Milk Dilutions with the Addition of Carbohydrates.—Diluting milk and adding sugar or some other carbohydrate is at once the oldest and the most extensively employed method of milk modification down to the present time. It forms the basis of what seems to us the most rational and at the same time the simplest method of procedure and will, therefore, be considered in detail as the method of choice in feeding the normal baby. In any method of feeding we have of course always before us the baby's natural food as an indication of its food requirements It is rarely if ever necessary to give more of any food element than is represented in human milk. In our ordinary dilutions however the protein and the salts are commonly in slight excess. As far as the protein is concerned there is no evidence that this is harmful provided it is so modified that it is digestible. The excess of salts seems to do no harm practically and tends to be equalized by the lesser utilizability as compared with those of human milk. It is commonly necessary to give less of the other food elements, the fat and the sugar, than is represented in human milk, both because the fat is harder to digest and to utilize, and the sugar is tolerated in less amounts in a cow's milk as compared with a human milk medium. In adapting to an infant a food so foreign and so different from its natural food several things further must always be born in mind. The protein —i.e.,—nitrogen requirements must always be covered. This is amply provided for in any dilution that is ever necessary in a practical feeding. The same is true of the salts in a practical way. The fats and the carbohydrates, whose functions in the economy of nutrition are very much alike, may replace one another within rather wide limits in practical feeding without perceptible nutritional harm.

The degree of dilution necessary to make cow's milk more acceptable to the baby has been determined purely empirically, always bearing in mind that the protein must not fall below that of human milk for any length of time. The amount of dilution commonly used varies considerably with different clinicians, and depends to a large extent upon other factors in the milk itself. If milk is boiled, or alkalinized, or citrated, it does not need to be diluted to the same extent as if it is given raw. In my own experience with the normal baby I rarely dilute the milk with more than an equal part of diluent. It is true for many years I have used only boiled milk (boiled two to three minutes in a sauce pan) and in beginning a cow's milk mixture for the first time especially in the newborn baby I commonly add for a time 2 gr. of sodium citrate to each ounce of milk in the mixture, or 1 or 2 oz. of lime-water to the day's food. Furthermore, I use as a routine a cereal water like barley water even in the newborn. Those who use raw milk, especially if they do not use alkalies or cereal waters, will find it safer to begin with dilution of one to two, or even three as is commonly recommended. Rationally, dilution has its basis also in the fact that one does not want to tax the infant's ability to utilize the protein by giving too great an excess, although the evidence of practical harmfulness of such an excess is wanting.

If, then, milk is diluted one-half for the newborn baby, one can gradually increase the strength of the milk at the expense of the diluent until toward the end of the first year the baby can safely be placed on undiluted milk. It is my own practice to reach this point at about the eighth to the tenth month—simply adding 2 or 3 oz. of water to make up for the loss due to evaporation in boiling. If raw milk is used it is commonly necessary to put off the use of undiluted unmodified milk till after the first year, or even well into the second year as is commonly advised in our text books. I have purposely omitted suggesting different strengths of food for different intermediate ages, and especially the use of tables. Tables tend to take the place of the baby. The food should be adapted very gradually to the individual baby's needs, not to a table.

By diluting the milk as indicated the protein and the salts that occur in about equal proportional excess in cow's milk are reduced to an amount more nearly approximating that of human milk, and to a safe degree. When the milk is diluted with equal parts of diluent the fat and the sugar, that are more nearly the same as in human milk, are reduced one-half—the fat to one-half and the sugar to less than one-third that of human milk. The natural procedure would then be to increase the fat and the sugar to the extent that the infant's tolerance will permit.

The *addition of fat* can readily be accomplished by adding a certain amount of cream, or by the use of top milk in place of whole milk. There is however a unanimity of opinion that cow's milk fat is not only hard to digest, but that it seriously retards emptying of the stomach in direct proportion to the amount contained in the food, and that it leads when given in excess to far-reaching metabolic disorders. For these reasons the fat is invariably kept low in amount, or in percentage, during the first few months of life. In a one to one dilution the fat is reduced about one-half, but as our mixtures are usually weaker than is human milk, i.e., of lower food value, we commonly give a larger total amount and this to some extent increases the amount of fat. Usually, too, by the end of the second month the dilution does not exceed one-fourth, or at least one-third, and this would bring the fat up to about 2.5 to 3 per cent. from that time on. That such amounts of fat as are represented in such dilutions as I have suggested are adequate from a nutritional standpoint, and the larger amounts are often not safe, has been amply proven by experience. The growth promoting *Fat Soluble A* is more than amply covered. In simple milk dilutions, then, the protein, the salts, and the fat are looked after by the mere dilution of the milk.

The *addition of sugar*, or other carbohydrate, remains to be considered. In a one to one dilution the sugar has been reduced to about 2 per cent. as compared with 7 per cent. in mother's milk. Nature's manifest demand for a large amount of fuel must be met to the full extent possible. Unfortunately it is commonly not possible to do the natural thing, i.e., add milk sugar up to an amount represented by the 7 per cent. of sugar in human milk. The dosage of sugar must always be graduated very carefully to avoid indigestion and fermentation, which arise only too easily in an artificial medium. It is one of the most constant considerations in artificial feeding to avoid these disorders at all hazards, because when they do occur it means not only a reduction of the sugar to a point at which the disturbance began, but a reduction not alone of the sugar but commonly of the whole mixture to a point far below that point. Two questions always arise when one starts to add sugar to a food mixture of diluted milk. What sugar shall I use, and second, how much?

The fact that *milk sugar* is the normal sugar of all milk, and that the evidence of the greater value of other sugars in the normal baby is not conclusive, together with habit, probably constitute the reasons why many of us use milk sugar at least in the first few months of life. Cane sugar is probably just as good, and cheaper; and there is certainly no objection to the use of dextrin-maltose preparations, and especially so in the normal baby where one wishes to give a large amount of sugar, and in abnormal, or borderline cases where there is undoubtedly a greater tolerance than for the other sugars. (See discussion of sugars elsewhere.)

The *amount of sugar* to use must be determined by the infant's tolerance, and also by certain data that are the result of experience as to how much sugar normal infants will usually tolerate in given mixtures. This can be explained in terms of ounces, or grams; or in terms of percentages of the whole mixture. The former is naturally more accurate as it represents a definite amount, while the latter is a variable amount depending upon the total quantity of food mixture. It is always well to begin with a small amount of sugar, ¼ to ½ oz. and to increase this amount gradually, a dram at a time, up to a maximum of ¾ to 1 oz. In terms of the kitchen one can begin with two to four level teaspoonfuls and gradually increase the amount up to 6 or 8 or 10 level teaspoonfuls in the total mixture. The dextrin-maltose preparations can commonly be used in much larger amounts, up to three or four tablespoonfuls. The amount of sugar that one uses depends to a considerable extent upon the composition of the mixture. If the milk is relatively small in amount, that is it is greatly diluted, then we can use more sugar than if the milk is greater in amount and more concentrated. The same relationship exists to some extent between the fat alone and the sugar. Likewise if the milk is boiled, or the casein eliminated by other means as a disturbing factor, a larger amount of sugar can be given than if the milk is raw and the casein unmodified except by dilution. Some clinicians will use much larger amounts of sugar than others for these reasons, e.g., one man will feed a five month old infant on half milk with 1½ oz. of sugar, another would feed the same baby with three-quarters milk and less than 1 oz. of sugar, with equally successful results. It is my own practice to give relatively much milk and relatively little sugar, rarely over an ounce of the latter. This gives the baby a relatively larger amount of fat than if the procedure were reversed.

The addition of *starch* to an infant's food is a valuable adjunct and meets two indications. Even the newborn infant can digest starch advantageously in such small amounts as are found in barley or oatmeal water. In the first few months however, cereal waters are used primarily as diluents because of their marked inhibiting effect on curd formation. It is for this latter effect chiefly, but also because of its food value, that I use cereal waters as a routine in younger infants. It is an interesting and common observation that an infant that will not gain on milk and water and sugar alone will at once progress more favorably if only a small amount of starch is added to its food. The fact that an equal amount of sugar, into which starch is ultimately converted, does not have the same effect has led to the hypothesis that the starch has some not yet understood effect upon the metabolism of the fats, carbohydrates, and salts, possibly through the fermentation products that arise from bacterial action on the starch in the intestinal tract.

This effect of the starches is especially noticeable when *after the third or fourth month larger amounts of starch are added.* There is no more striking and certain effect in all infant feeding than that which results when an infant of five months, or even four months, that has not done well on milk and water and sugar gets an appreciable amount of starchy food. If it seems desirable to add this larger amount of starchy food to the bottle it can be given in the milk in the form of wheat, oatmeal, or other flour; or in the form of flour ball, or Imperial

Granum. It is my own preference to give this food *from the spoon* in the form of farina, or cream of wheat, or, after the fifth or sixth month, oatmeal, for reasons that will be explained later. (See additional food during the first year.)

Toward the latter part of the first year the diluent is gradually lessened as the milk is increased; the sugar and starch are gradually eliminated and the baby is on undiluted whole milk, which should never exceed one quart a day.

Having decided in a general way about what degree of dilution seems desirable, together with the amount of additional carbohydrate that seems indicated, it is necessary to approach the more important subject of the total amount of food to give in the 24 hours. This is so intimately interwoven with the question of the number of bottles to give in each 24 hours, and also the amount of each feeding, that these will be considered first.

Feeding Intervals.—It has always been taught and is still taught in many of our leading text-books that the newborn infant should be fed every two hours for the first one to eight weeks. Some of our other text-books advocate feeding all babies every four hours, giving only five feedings a day, and this even in the premature baby. In Germany the four-hour interval, following the lead of Czerny and Keller, has become the rule. That such difference of opinion can exist among equally experienced and equally successful clinicians can admit of only one explanation—that the baby can be fed every two hours, or every three, or every four hours with nearly equal success, and that most of us follow the old path from habit rather than try a new one. When one bears in mind, however, that babies vary greatly both in health and in sickness, one cannot help but wonder whether in foisting a fixed interval varying from two to four hours upon them one is not training them as acrobats to perform certain stunts rather than treating them as normal individuals who have varying tastes and temperaments, to say nothing of varying stomachs and states of morbidity.

Before "regular intervals" were established babies were nursed or fed from the bottle whenever they cried, or seemed hungry. That regularity and a long interval are desirable was recognized even by Metlinger[3] (1473) when he recommended feeding intervals of "over one or two hours." The two-hour interval came to be looked upon as a normal one all through early infancy and was a distinct triumph over earlier practices. Theoretically, too, the two-hour interval seemed to rest on a firm basis at least in the newborn baby. Earlier attempts to measure the stomach capacity of the newborn baby led to the conclusion that it held from 1 to 2 oz. varying with different babies. If the stomach capacity was to be taken as the measure of the individual feeding, as seemed reasonable, then it was manifestly necessary to feed the baby as often as every two hours in order to make the food intake equal the known requirements of the infant. It soon became evident, however, that the capacity of the stomach as measured in the baby after death, and even the capacity of the living, elastic and distensible stomach, was not the true measure of the amount of liquid food the baby could take at one feeding. It was found that the pylorus was relatively wide in infancy and the muscular control relatively incomplete as compared with the adult. When a baby takes a certain amount of liquid food some of it passes beyond the pylorus while the latter part of the feeding is entering at the cardiac end of the stomach. This is accompanied by little if any danger of over-distention for this reason itself, and besides the baby would vomit or quit nursing before such danger was imminent. Only with the use of the stomach tube would one hesitate rapidly to ingest an amount of food that exceeded to any extent the known actual capacity of the stomach. That the total digestion is not reduced by the fact that some of the food passes on prematurely would seem to be evident from the fact that all digestive functions are active in the intestine and to a far greater extent than those found in the stomach, with the possible exception of the coagulating ferment, which is however also found in the intestine. The question of the amount of food that can be given at each feeding, and this carries with it the question of feeding intervals, can therefore safely be left to other considerations than stomach capacity.

In recent years, following the lead of Czerny and Keller,[14] who advocate feeding all babies, except in certain pathologic conditions, every four hours, and only five times in the 24-hour period, there has been an almost universal tendency to longer feeding intervals. The two-hour interval that was at first considered the proper one, especially in the breast fed baby, had, of course, long before this been lengthened by common consent to three hours after the first few months, and to four hours toward the latter part of the first year, and that especially in the bottle fed baby. This was based partly on empiricism, partly on the known larger capacity of the stomach of the older infant, and partly on the observation that the stomach of the cow's milk fed baby required a longer time to empty than that of the breast fed baby. From a theoretical point of view the last consideration, the emptying time of the stomach, was naturally pushed into the foreground. The statement became current that the emptying time after a normal breast feeding was about two hours, after a cow's milk feeding about three hours. This was accompanied by the further statement that the feeding interval should be longer than the emptying time, to permit of a period between each two feedings during which the stomach would "be rested," and also to permit free hydrochloric acid to be present to exercise its germicidal action on the stomach contents, and its remoter action on intestinal digestion. Unfortunately definite statements on the emptying time of the stomach in the artificially fed baby are not yet possible. This is partly due to the fact that sufficiently accurate data are wanting; but, even more important, the emptying time depends upon so many factors that bear upon the subject that a blanket statement never will be possible. The effect of the fat and the physical state of the casein upon the emptying time has been considered in detail in preceding pages and needs only to be mentioned here. In my own experiments I was constantly impressed with the fact that the stomach seemed to empty more slowly than was commonly thought, especially when cream or raw milk were used. If a perfectly normal adult fails even approximately to empty the stomach within five hours after taking a pint of 16 per cent. cream, or a quart of raw whole milk, amounts that are not relatively excessive as compared with those taken by an infant; then it seems probable in the absence of data to the contrary, that the emptying time in the infant is greater than is commonly held to be the case, unless the fat is kept low and the casein adequately modified. In general, I think one could safely venture the statement that with ordinary cow's milk mixtures the emptying time of the infant's stomach is much nearer to four hours than to two hours. The greater tendency of the bottle baby to vomit, as compared with the breast baby, probably finds here its greatest reason. Ultimately again, as everywhere else, theoretical considerations give way to the baby himself. The baby tells us quite unmistakedly that as a rule with a longer interval he is more comfortable, sleeps better, has less indigestion and fermentation, and finally can take more food safely than if he is fed more often. The comfortable baby will be less spoiled with all that that means both in infancy and in later life. The mothers too appreciate even more than the babies the benefits in many ways that come from longer intervals. The baby however reserves to himself the privilege of being an individual and of having something to say about how often he as an individual shall be fed.

That normal babies will commonly thrive comfortably on a four-hour interval even from the very start has been amply demonstrated. The same is true of the three-hour interval and I am not sure that it is not of the two-hour interval. If the newborn baby can "thrive comfortably" on a two-hour interval then surely the older one can, though other considerations make such an interval wholly undesirable. Dogmatism limits vision, obstructs reason, promotes dissension, and often arrogates to itself the place of authority. It has been an obstacle of the first magnitude in the evolution of infant feeding. To say that every baby, even the premature, the sick, and the marantic, should be fed every four hours as some extremists are doing as if this number had been inscribed on the Tablets handed down from Mount Sinai, is to my mind as pernicious as it would be foolish to say that every baby regardless of age, or health, should be fed every two hours. Early in my practice I fed babies at conventional short intervals. For a period of years I surrendered unconditionally to the four-hour interval. I now feed a baby at whatever interval I think that individual baby should be fed. For didactic purposes only the situation must be stated more definitely, and can be summed up as follows: It is rarely necessary or desirable to feed a normal baby oftener than every three hours. This does not include the premature baby, or the "lazy" puerperal baby which should be fed oftener. For many normal babies the four-hour interval is not the optimum during the first few months and is commonly not the best interval in the first month or two. It is my own practice to begin the normal newborn baby on a three-hour interval and as quickly as seems advisable from the baby's and every other standpoint to change to a four-hour interval. This is commonly some time in the first three or four months, but is often deferred until the second half year. I find myself commonly in the position of having to ask the mother how often we are feeding the baby at the present time. Fractional intervals such as two and a half or three and a half hours rarely seem necessary or indicated.

One of the manifest objections to the "four-hour interval, five feedings a day" program lies in the inability to give enough food in the 24 hours without giving a larger amount at each feeding than the baby will take. I have before me a well known text-book that recommends for a baby of two months, weighing 10 lb., a little more milk than water and from ¾ to 1 oz. of a malt food, to be given in five feedings of 4½ oz. each. The energy quotient of this baby would be from 67 to 79 although the author elsewhere subscribes to the commonly held idea that a baby at this age requires about 100 calories. To meet the latter requirements the two months old baby would need to take of this mixture 5½ oz. at a feeding.

The number of feedings in the 24 *hours* to be given the newborn infant, if fed every three hours, is commonly seven. This would allow for three-hour feedings during the day and longer intervals at night. The length of the night interval can commonly be left to the baby, provided the period is not too short. Even here it is sometimes well to compromise and let the baby have it every three hours for a time rather than to keep the mother awake unnecessarily. If it will sleep longer at night than planned for this is of course desirable unless the number of feedings falls off to such an extent that the baby cannot get enough in the remaining feedings. If this should happen the baby must be awakened or else the total food must be put in a less number of bottles. Some time after the first month and sometimes earlier, the feedings are naturally reduced to six, and can then be given either at four-hour intervals day and night, or at three-hour intervals during the day with only one feeding during the night. This one-night feeding can be given either at 9 or 10 p.m., or when the baby naturally wakes up for it, at the mother's discretion. Toward the middle of the year five feedings will usually be found ample, then nearly always at four-hour intervals, with one feeding again at 10 p.m. or when the baby wakes up. At one year, often earlier, four feedings are enough and during the latter part of the first half of the second year the child can commonly be put on three feedings a day.

Shall the Baby Be Awakened for Its Feeding?—This question cannot be answered categorically. In general it would seem more natural and wiser to let the baby sleep beyond its feeding time cutting down the succeeding interval, if the baby is awake, so as to get around to the established routine and hours again, that are of more value to the household than to the baby. If the baby shows an inclination to sleep beyond the allotted interval as a rule, then the interval should, of course, be lengthened to an extent that would naturally eliminate this question. Common sense would sometimes dictate feeding the baby a little short of the established interval if it seems unusually hungry. I know of no better *reductio ad absurdum* of the ultra modern slavish adherence to fixed hours of feeding than the custom of setting the alarm clock for the night feedings; or the tired mother walking the floor with a crying baby waiting an hour for the clock to announce the time for another feeding! There is, however, a type of baby that is not uncommon that will sleep all day and wake up every few hours at night. Here the baby should be fed at regular intervals during the day regardless of sleep, and allowed to sleep as long as possible at night.

While *in the hospital*, where many of our fixed ideas originate, it is necessary to keep babies to regular hours from the start for routine reasons. In the home where everything revolves about the baby and especially in the first few months it is often better at first to sacrifice accuracy to discretion, gradually leading the baby along a path of less resistance to more regular habits. In determining a course to pursue the practical physician will often judiciously weigh in the balance the opinions and preferences of the mother and even of the grandmother whose memory reverts to a time when babies were reared with apparent success without the aid of a clock!

The length of time of each nursing from a purely digestive standpoint is of no practical interest, and can usually be safely left to the baby himself. It is now known that milk coagulates more coarsely in the stomach if the food is taken slowly and from that viewpoint alone one can only favor rapid nursing.[7] There are other considerations however. The stomach always contains air, the gas bubble, or Magenblase of the roentgenologist. If the milk is taken too rapidly to allow of relief of pressure through some of the milk leaving by way of the pylorus, the gas bubble is unduly compressed and leads to discomfort, or sudden violent belching with accompanying regurgitation of food. It is probably for this reason that too rapid feeding leads to vomiting. It is of practical value to be familiar with this fact and its remedy. Many babies must belch up this gas bubble after every feeding, or even during a feeding, before they are comfortable or can resume nursing. If they are in the erect position so that there is no food between the gas and the lower esophageal opening the gas alone escapes. If the child is not in this position the gas brings food with it, often with a decided explosion. After each nursing, or if uncomfortable during nursing, the baby must be picked up and held in the nearly erect position by holding it over the shoulder and gently pressing the stomach against the shoulder, or patting its back. This "patting up the bubbles" can also be accomplished by sitting the baby erect and gently pressing the stomach between a supporting hand in front and one in the back. The common admonition to lay the baby down gently after nursing so as to prevent regurgitation is of little value in many cases unless the gas "bubbles" have first been "patted up." In general a feeding should take from 10 to 15 minutes. If the hole in the nipple is too large the food often fills the mouth more quickly than it is emptied and leads to choking and sometimes to vomiting from this cause.

Amount of Each Feeding.—From all that has been said it becomes evident that it is not of great importance to carry certain figures in one's head to represent the amounts of food that babies should get at each feeding during different months of life. The important thing is the amount of food given in the 24 hours, the degree of dilution is next, and the amount at each feeding is least in importance. In general the newborn baby, fed every three hours, can be offered what he will take of 3 oz. at a feeding. Commonly he will take less at first and by the time he will take 3 oz. it is not too much. This amount is gradually increased until about the sixth month when an 8 oz. feeding is not too much and this amount need rarely if ever be exceeded. Combining all the factors that enter into a mixture it might be stated in another way that we begin with a little over a pint and gradually run up to a quart or a little over a quart. Practically, one commonly decides first upon the number of bottles to give; then, upon data to be discussed presently, on the amount of milk and sugar one wishes to give in the 24 hours food; one adds to this an amount of diluent that will give about the degree of dilution and at the same time the total bulk of food desired; and then divides the total amount of food by the number of bottles to be given. For example: A baby two months old weighing 9 lb. we decide ought to have six feedings; it ought to be able to take about 16 oz. of milk; this we would dilute with say 8 or 10 oz. of barley water. This would give us 24 to 26 oz. of food to be divided in six feedings, or 4 to 4+ oz. each. With a little experience one naturally adds the diluent up to some round figure, that when divided by the number of bottles will give a round number of ounces, so that small fractions may be avoided. As one becomes more experienced consideration of the number of bottles and the amount in each become inseparable and usually represent the first step.

The Total Daily Quantity of Foods.—The total quantity of food given in the 24-hour period, provided always that it is well balanced by a proper representation of the different food elements, if of greater importance than the degree of dilution, the total bulk of food, the amount in each bottle, or the number of bottles. If the baby gets too little it will not gain. If it gets too much it will either gain excessively or will show some digestive or metabolic disturbance. Overfeeding in general, with one or the other of the food elements, is the one great factor in the causation of the nutritional disturbances in infancy. Practically, it is synonomous and coextensive with the pathogenesis of what we ordinarily mean by nutritional disorders of infancy. The prevention of overfeeding is, therefore, always uppermost in our minds in feeding babies. To Biedert we owe the inception of the idea that an infant should get the minimum amount of food (Minimalnahrung) on which it will thrive normally. While the optimum amount may be a little higher than the minimal amount for all practical purposes they are indistinguishable. Two factors enter into an estimation of whether a given baby is getting the proper amount of food: *First, foremost, and all the time, the baby himself;* and, secondly, certain numerical standards that represent the amount of food that we have found empirically and experimentally to be necessary to insure normal growth and nutrition in the average baby. The former is always the ultimate criterion, the latter is simply a check or control on overfeeding and underfeeding that is of greatest value to the beginner.

The careful observation of the baby himself is the *sine qua non* in the successful feeding of the individual baby. It never has been and never will be superseded by any other standards, valuable as these may be as helpful guides. All babies of the same age and weight and apparent degree of normality do not require the same amount of food. It is a matter of common knowledge that of two babies of the same age and weight and with no evidence of digestive disturbance one will develop normally and the other will present a florid rickets. The criteria by which we judge normal development will be considered in detail later. The two important tangible items *with reference to the amount of food* are the gain in weight and the evidences of indigestion. If the baby does not gain in weight and has no evidence of indigestion it commonly does not get enough to eat. If it gains normally, the food is well balanced, and there is no digestive disturbance then the baby is getting enough food and not too much. If there is a digestive disturbance this alone means that the food is exceeding the baby's present tolerance. Fortunately, the more serious disturbances are nearly always preceded by easily recognizable milder symptoms, the most important of which are vomiting, a tendency to diarrhea, or looseness of the bowels, scalding of the buttocks, and discomfort. *To watch for these milder symptoms, to recognize them, to make the proper deductions,* and to *remedy the condition while it is still easy to do so, these are the important and indispensable factors in the prophylaxis of the more serious food disorders.* An excessive gain in weight such as 8 to 12 oz. a week, while not necessarily calling for a reduction in the amount of food, does call for added vigilance, for such a gain in the artificially fed baby usually means overfeeding and its attendant dangers. Only by careful observation of the baby itself, furthermore, can the development of remoter evidences of dysnourishment such as anemia, anorexia, rickets, and scurvy, be anticipated.

Numerical Standards, or Guides.—The beginner will find of very great practical value certain numerical guides that will be of great help in preventing overfeeding and underfeeding on the one hand, and on the other hand in estimating, while he is gathering his own experience, whether a given food meets the usual well-known nutritional requirements of the average normal infant. The sick infant is in a class by himself, the healthy infant runs constant enough so that such a guide will help to make up for lack of experience. The experienced feeder no longer needs such guides, or rather he uses them, or others of his own, subconsciously. Two such guides, or controls, naturally suggest themselves when using such mixtures as have been described, one based on the number of ounces of milk per pound of baby, the other on the number of calories per pound or kilogram of baby.

Milk Volume Control.—When using such dilutions and modifications of milk as have been suggested it has been found more or less empirically that the amount of milk in the 24-hour food mixture usually taken by healthy, thriving infants lies between 1½ and 2 oz. to the pound of baby. If one uses much sugar in the mixture, say from 1 to 2 oz., then it is commonly not safe to exceed 1½ oz. of milk per pound. If, as is my own practice, one rarely exceeds an ounce of sugar, then it is safer to approach more nearly 2 oz. per pound. Thus, if a normal baby of 12 lb. is getting about 18 oz. of milk in its food with 1½ oz. of sugar; or about 22 oz. of milk with ¾ oz. of sugar, and is thriving normally we would feel that it was getting about the right amount judged by this standard. It will be found that both babies are getting about the same amount expressed in calories, and that this amount corresponds to its known caloric needs (about 100 calories per kg.). The infant's protein needs are amply covered by 1½ oz. of milk per pound. This guide in feeding should, of course, be used only as a guide, or check, never as an advance measure of the amount of food to put a given baby on regardless of the clinical aspects. Thus used it becomes for this purpose the most common and most practical aid in artificial feeding when simple milk dilutions are used.

Caloric Control.—While such a standard as has just been described is of great simplicity and of great value when simple milk mixtures are used it is of no value with formulæ of different construction. The calorie is the universal measure of food value and alone can be used under all conditions. To Rubner and Heubner we owe the practical application and popularization in infant feeding of the caloric needs of an infant as a measure of its nutritional requirements. From a number of observations on normally thriving artificially fed infants, under conditions unexceptionable for such a study, Heubner[33] determined that infants of the same age and weight required very much the same relative amount of food, and that this amount steadily diminished with advancing months. He expressed the amount of food taken in terms of calories and the number of calories per kilogram of body-weight of baby taken daily as the *energy quotient.* He found that this energy quotient in the cases studied "from the third week of life till about the end of the sixth month was 100; in the later months till toward the end of the first year this amount gradually diminished."[34] The energy quotient necessary to maintain a stationary weight during the first six months he found to be about 70. Since these observations were made a great many reports have appeared from numerous observers on the amount of milk normally consumed by healthy thriving breast fed infants weighed before and after each nursing for months. In all of these the energy quotient was practically the same as that of Heubner's artificially fed babies, except that in some it was constantly higher, in some constantly lower. Schlossmann and others were inclined from their own observations to put the energy quotient at 110 during the first three months. Czerny called attention to cases that progressed normally on considerably less than 100. Since this time a very extensive literature has centered about this theme with views varying within such small limits that they only emphasize the great value of Heubner's conclusions.

In this country the whole procedure has been given a very belated and rather begrudged recognition, until recent years, especially among the advocates of percentage feeding and that for obvious reasons. It seemed quite unnecessary, if percentages adequately expressed food values, to add this to the calculations already necessary, moreover, it came intimately associated with the use of simple milk mixtures which were not in favor and were looked upon as alien in origin. Some of its more ardent advocates unfortunately too raised it to the position of a rival "*Caloric Method of Feeding,*" as compared with the "Percentage Method." It has even been urged ever recurrently that a calorie is merely a measure of energy not of food value, that an appropriate energy quotient could be supplied with sugar and water alone as if the practical employment of the energy quotient as a measure of food value in infant feeding per se set aside all ordinary intelligence!

The figures of Heubner, if used as *intended*, can safely be used as a valuable general guide, but are in no way to be considered as fixed amounts. If an infant gains properly on a smaller amount so much the better; if it fails to gain on an energy quotient of 100 and shows no clinical evidence of overfeeding then one serenely advances to a point where it will gain, regardless of energy quotient. The active baby needs more food than the phlegmatic baby, even up to 40 per cent. more. The question naturally arises: "Why bother with it at all if the ultimate criterion is the baby?" I can only revert to my earlier years when I fed babies with only percentages and tables to guide me, in addition to what little I could glean from the baby himself, and I can truly say that at that early period I followed a rudderless course. In a paper[34] published some dozen years ago I reported the results of a retrospective investigation of my earlier practice along these lines. I found that I, and the same was apparently true of others, had commonly fed babies with energy quotients up to 150 or 200 and even 280; and occasionally babies with a complicated formula that seemed nourishing enough were kept for weeks on only 50 or 60 calories. The outstanding fact is this and we are apt to forget it later: It takes years of observation to get to a point where the baby alone is a safe criterion. The beginner who has these years before him, or is in the midst of them, needs something more to guide him during that period. The evidence of this is about us every day. Only after years of experience can we gradually lay aside, or rather absorb, these standards, and use the baby as our sole guide. Even then every mixture that we prescribe is only the subconscious, or conscious, expression of what was once very conscious and very concrete. The experienced reader no longer spells or "sounds" his words, but the letters and the sounds that were once so real are still there though he is not aware of them.

The beginner will do well literally to figure out the energy quotient of every feeding *after he has written it.* It is to be hoped that he has been taught never to use it as a measure of what to give a given baby, but rather as a control, or check, on what the baby is getting. Unfortunately there is still an impression about that there is a "Caloric Method of Feeding Babies." The *reductio ad absurdum* is evident from the following instance. A young physician who had only recently left the hospital was called to see a young infant. He inquired about the age and weight, took out a pad and pencil, figured out how many calories the baby needed to represent an energy quotient of 100, translated this into a judicious mixture of milk, water, and dextri-maltose, divided this by five and handed it to the mother with instructions to feed every four hours. The item of not even looking at the baby, nor inquiring about its stools, to say nothing of examining them, evidently made more of an impression on the mother than it did on him judging from her subsequent action.

After a little experience the necessary calculations can be made without paper and pencil in a few moments. After a time the energy quotient can be estimated very closely in simple mixtures from a mere glance at the formula. The experienced feeder easily figures out the energy quotient—at a glance he can tell from all his past experience that a mixture he has just prescribed conforms accurately both as to relative volume of milk, and within a few points as to energy quotient, with what he would want the baby to have. Hair splitting accuracy is as unnecessary as it is unreasonable.

The *calculation itself* is very simple. It is necessary to know the caloric equivalent of 1 oz. of each of the ingredients used in the mixture on the one hand and the baby's weight on the other. (Table of caloric values.) If now a baby of four months that weighs 12 lb. is getting a mixture of 20 oz. of milk; 10 oz. of barley water; 1 oz. of sugar, the energy quotient is determined as follows:

Milk........... 20 oz. = 20 × 21 c. = 420 calories
Barley water.... 10 oz. = 10 × 2 c. = 20 calories
Sugar.......... 1 oz. = 1 × 120 c. = 120 calories
Baby's weight 12 lb.)560 calories
46 + calories per pound = energy quotient
2.2
92
92
101. + calories per kg. = energy quotient.

The *energy quotient* can be expressed either in calories per pound or calories per kilogram. In European countries the metric system predominates and, therefore, the latter is commonly used. In changing from one to the other one must either multiply or divide as the case may be, by 2.2, the number of pounds in a kilogram. The energy quotient expressed in calories per pound that would correspond to 100, 80, and 70 would be approximately 45, 36, and 32.

Table of Approximate Caloric Values of Common Foods

	Ounces	Calories
Human milk	1	24
Cow's milk	1	21 (up to 25)
Cream (16 per cent.)	1	54
Fat free milk	1	11
Butter milk	1	10
Eiweissmilch	1	12
Dried milk	1	127
Evaporated milk	1	50
Condensed milk	1	95
Malt soup (Keller's)	1	24
Malt extract	1	90
Sugar	1	120
Dextrin-maltose preparations	1	110
Flour	1	100

In feeding a baby on cow's milk for the first time it is always best to begin with a smaller amount and gradually under clinical control proceed to the desired amount. *Advance slowly, retreat rapidly* is a good dictum in infant feeding that knows few exceptions. The first attempts are always tentative, one feels one's way along to see how the baby will react. If one goes too fast and the baby's digestion is disturbed it becomes necessary to retreat to a point some distance back of the point at which the disturbance occurred and this inevitably entails a loss in weight. It is a cardinal principle in infant feeding to proceed so slowly that one never has to retreat. Beginning with the smaller amount one advances the milk an ounce at a time and the sugar a dram at a time rather than to make such jumps as from one-half milk to two-thirds milk, or from ½ to 1 oz. of sugar. A practical way of proceeding in such a case can best be illustrated by an example. A normal healthy baby four months old, weighing 12 lb., is to be fed wholly artificially for the first time. Five feedings of 6 oz. each to be given at say 6, 10, 2, 6, 10 would seem an appropriate amount for the daily food. I commonly write out for the mother some such schedule as the following beginning with an amount that I feel sure the baby will be able to handle.

Milk	15–16–17–18–19–20–21–22 oz.
Barley water	15–14–13–12–11–10– 9– 8 oz.
Sugar	4 level teaspoonfuls gradually increased to 6 or 8.
Sodium citrate	20 gr.–20–20–20–20–20–20–20 gr.

5 feedings, 6 oz. each, feed every 4 hours, or
6 feedings, 5 oz. each, feed every 3 hours.

Boil milk 2 or 3 minutes.

The mother, or nurse, is then instructed to begin with the first vertical formula, milk 15, barley water 15, sugar 4 level teaspoonfuls, and sodium citrate gr. 20 and to advance the milk an ounce a day, or every two days, until she has reached the last formula (milk 22 oz., etc.). The barley water is diminished in proportion as the milk is increased. The sugar is cautiously advanced every few days, a teaspoonful at a time, under careful clinical control, and not to exceed a total of 6 or 8 level teaspoonfuls. When the last formula is reached and all is well for a few days the sodium citrate can gradually be removed, again under clinical control, over a period of a week or so. While the sodium citrate is perhaps often not necessary it nevertheless adds a degree of assurance in my experience that I do not like to deny the baby, nor myself. One or two ounces of lime-water gives practically the same result. The kind of sugar is of course specified.

The mother is instructed to boil the milk if this seems desirable for two or three minutes actively in a sauce pan and to supply additional water equal to the amount that has evaporated. The baby is best fed every four hours, unless he has been fed every three hours hitherto and the mother prefers to put off the change for a time. The food can be divided into five feedings of 6 oz. each or six feedings of 5 oz. each to meet this alternative.

The mother is warned that the baby will probably not gain the first week, may even lose a little for a few days, but that he ought to gain the second week and thereafter. After the last formula is reached, she is told, the baby is to be kept on this till he no longer gains 4 or 5 oz. a week. This last formula always represents what I think the baby ought to be getting now, it comes up to the standard of having nearly 2 oz. of milk to the pound of baby, somewhat less than 1 oz. of sugar, and from past experience I know that such a combination gives an energy quotient of about 100.

The mother is told finally that on such a food the baby will not have the soft, mushy, acid gold-yellow stool it had when on the breast; that he ought to have one, or at most two, formed, or at least pasty, smooth, offensive, pale-yellow stools; and that if he has more, or has even a single very soft, frothy, or acid stool or one that irritates the buttocks, she must report that fact at once so that the condition can be remedied quickly by an immediate reduction of the food. If progress is unimpeded she can report once a week for a time.

Additional Food during the First Year.—There are still rather wide variations in the opinions of different men as to other than bottle food during the first year. Among the laity and among practitioners the idea still exists that one must be very cautious about giving any other food than the bottle throughout this period; and especially so in the case of the baby that has not done well on the bottle alone and during the summer months, the spectre of which still haunts us without much reason. Nearly all are agreed that orange juice at least should be given during the second half year if not before. So far as I know only one authority advises no orange juice as a routine and no food but the bottle during the whole first year. To my mind such teaching is not only not harmless, but positively harmful and that for several reasons. In the first place it is a matter of common knowledge that orange juice is not only a powerful, but from every standpoint the most acceptable, anti-scorbutic that we possess and that no matter what artificial food we give some such agent is indicated to supplement the anti-scorbutic element in cow's milk which is none too strong even in raw milk. I have yet to see any unfavorable effect from the use of orange juice if given judiciously yet adequately, after the second or third month, except that I am convinced that it makes the occasional baby vomit either the orange juice or the next feeding or both. It is also conceded that babies that get only milk till toward the end of the first year are commonly pale, flabby, and rachitic, for reasons detailed in preceding pages. This leads to my second reason and carries with it a brief consideration of a very important and much neglected phase of infantile psychology.

Babies form habits that, like all habits, are easily broken at a certain time but are hard or even impossible to break if they become too firmly fixed. This is notably true with reference to methods of taking food. The baby that has nursed exclusively for three months or less will practically always take a bottle with very little trouble. At the fifth or sixth month the transfer to the bottle is commonly very difficult if the baby has not been accustomed to a bottle, sometimes it can no longer be accomplished. At nine months the effort to induce the baby that has never had a bottle to take it is well nigh hopeless, and shortly after this wholly so. The obverse is equally true as we find when we attempt at various ages to switch a baby from the bottle to the breast. Starving the child into taking the bottle, or the breast as the case may be, does very little good, and forcible measures none whatever, for the baby does not recognize the new thing offered as food.

In a large measure the same thing is true of the spoon food as opposed to the bottle or breast. The baby that has had no spoon food until he is a year old, in my experience, has a hard time convincing himself that what is offered to him is food, or that it is as good a way of taking it as he is accustomed to. Very commonly he will finally consent to take a limited number of things that happen to appeal to him, and he becomes one of those picky and fussy individuals who "can't eat this and never eat that." I recall vividly a baby of 13 months that was still nursing a rapidly waning breast and had been accustomed to no other manner of taking food. He never would take a bottle, and for days refused anything from a cup, a spoon, or even his hand. The mother's milk disappeared rapidly, and the baby lost 3 lb. before he could be induced to take a maintenance diet. For weeks he would eat only oatmeal, which seemed to strike his fancy, and oatmeal with which other things were incorporated to the extent that he would permit. He remained seriously undernourished and set against all but a few articles of diet for a year afterwards when he escaped from my notice. The child seemed normal physically and mentally in every other way.

This is doubtless an extreme case but I am not sure but that it was extreme only because of the rarity with which one sees a child so old that has never been given other food. There is much less trouble if spoon food is begun at the eighth or ninth month. I am myself firmly convinced that it is a serious mistake not to begin spoon food in the first half year of life, certainly not later than the sixth month, preferably at the beginning of the fifth month, or in the case of the baby that is not doing well on the bottle alone and has no tendency to diarrhea, at the fourth month.

The third reason is that *babies stand these additional foods well* with almost no exceptions. Their ability to digest starch is ample. If there was any doubt left about that the recent success with feeding boiled milk with 10 per cent. farina to very young infants with pyloric stenosis would seem to tend to dispel it. Orange juice never causes trouble except the occasional vomiting. Broths and vegetables are well borne, the latter if finely divided by mashing and running through a sieve. Toast, zwiebach, and crackers soften and liquefy rapidly. The very physicians who will not tolerate such "solid food," will for safety's sake give only "liquid" food such as raw milk that is the solidest food ever offered to a baby!

The time and manner of giving these additional foods is often a domestic problem purely—the important thing is to give certain amounts during the day, and always only at a regular feeding time, the only exception to the latter being the orange, or other fruit juices, which are commonly given between meals, for reasons that are traditional rather than based on demonstration. It is rather well in this matter to develop a certain feeding routine for healthy babies with normal tolerance for food for convenience sake. My own tendency for years has been to slide the whole sequence of additional foods farther and farther down the sale of months, always only because babies seemed to do better if I began still earlier than I had done. My own normal routine of the past few years is here given, not as a better one, nor as one that seems especially desirable to adhere to, but simply to furnish a concrete illustration of such a basic routine that can be varied to any extent that seems indicated. About the third month, sometimes earlier, I begin with a teaspoonful of orange juice about one hour before a feeding, commonly the 10 o'clock feeding. This can be given straight if sweet, or sweetened with a little cane sugar, or diluted with water to any degree and sweetened if necessary, according to the baby's taste, and is given either from a spoon or a bottle. This is gradually increased up to about an ounce at the fifth month, and to 2 or 3 oz. toward the latter part of the year. It is perhaps not necessary to say that orange juice is not given as a laxative, as so commonly held by the laity, for it has that property to so slight a degree that it is practically negligible. Prune juice can be given in about the same amounts at the same ages, purely as a laxative, but in no way as a substitute for orange juice.

At the *fifth month*, often earlier if the baby is not doing well and has no evident digestive disturbance, sometimes later if things are flourishing, the baby gets a tablespoonful of well cooked (20 to 30 minutes) farina or cream of wheat just before the 10 a.m. and the 6 p.m. bottle. This cereal is put in a dish, some of the bottle is added to it, a little cane sugar is added if necessary, and after the baby eats this *from a spoon* he gets the rest of the bottle. During the next few months the cereal is gradually increased through two or three tablespoonfuls to a regular dish at the seventh to the eighth month. After a month or two of these cereals oatmeal, rice, or other well cooked cereals can be substituted if it seems desirable and toward the latter part of the first year I see no objection to the occasional use of dried cereals such as corn flakes, puffed rice or wheat, and shredded wheat. It has never seemed necessary to strain the oatmeal or other cereals.

At the *sixth month* a *vegetable soup is added* to the 2 p.m. feeding. This is made up of a pound of beef or other meat, and a pint of water, to which is added a carrot, a potato, a small handful of spinach and some celery, all cooked together for about two hours down to 8 oz. The meat is removed, the broth and vegetables are passed through a fine sieve, and this thick puree is given in increasing dosage beginning with 1 or 2 oz. and rapidly increasing it up to 4 or 5 oz. by the seventh or eighth month if they are well tolerated. With this can be given crumbs of well dried zwiebach, or toast, or cracker in the soup, and shortly after this time zwiebach or dry toast in his hand to be eaten by still another method. A piece as large as two or three fingers will commonly disappear in a surprisingly short time whether teeth are present or not. The bottle immediately follows the soup, or in a short time, as a larger amount of soup is taken, this bottle can be lessened or even discontinued and the amount of milk thus eliminated can be added to the other bottles, especially to the morning and evening bottles, that are unaccompanied by other food. If the vegetables are finely divided by being sieved, or adequately mashed, they rarely give rise to any trouble at this age. Neither have I found that potato starch is peculiarly dangerous, or that beef broth is less digestible than mutton or chicken broth. I have myself never seen any clinical evidence of the correctness of either of these views, and the latter certainly wants demonstration though amply backed by tradition. A word of caution must always be given to the mother concerning vegetables. Spinach and carrots are never completely digested, or liquefied, under any conditions. The mere fact that recognizable remnants appear in the stools is no evidence of indigestion and no indication to discontinue them unless there is at the same time a tendency to diarrhea or looseness of the stools.

At the *eighth* or *ninth month apple sauce*, or baked apple, is commonly added to the evening meal. About the ninth month I suggest two alternatives that can be left to the discretion of the mother—either to continue the vegetable soup up to 5 or 6 oz. a day at the 2 p.m. feeding, or else to give a clear broth of the same amount with baked potato and spinach or carrots as a separate dish. About this time puddings of rice, tapioca, bread, etc. are permitted at the 2 o'clock or the 6 o'clock feeding. A piece or two of crisp bacon can now be taken from the hand, or else finely mashed and incorporated in some other dish. I am inclined to think the value of bacon is overestimated and that its popularity dates back to the thick, half cooked, greasy rind that was supposed to have an anti-rachitic property.

At the *end of the first year* the feedings are commonly reduced to four at four-hour intervals. Nothing should be given between meals, except perhaps the orange juice, not so much perhaps because the food would be harmful in itself, as because it leads to bad habits and to greater difficulties in discipline that may have a lasting effect. The baby is now taking about a quart, sometimes less, never more, of straight whole milk divided among the feedings as seems to fit best domestically. He gets vegetables such as well cooked and finely mashed spinach, carrots, baked or mashed potato, cauliflour, string beans, celery, thoroughly cooked cereals such as oatmeal, rice, wheat, cornmeal; dry cereals as a variation; broth, or if preferred beef juice; zwiebach, toast, soda or graham cracker; puddings, bacon, a bone with a little meat on it, orange juice, baked or scraped raw apple, or apple sauce, stewed prune pulp, finely mashed ripe banana; all at the regular feedings so divided among them as seems best for the particular baby in the particular household, provided he gets a proper variety as indicated.

There are both *advantages and disadvantages in variety of food.* If there is too little variation the baby either tires of his food which the perfectly normal baby is not so apt to do, or else he gets so fixed in the habit of liking only certain foods that he will not eat the variety of food with which later attendance at the family table brings him in contact. This is well illustrated in those children whose over-careful mothers or nurses will not allow them to have anything but mushy or most finely divided or sieved food, even into the second year. Often it becomes almost impossible to get such children to eat any coarser food. If on the other hand as so often happens when over-attentive mothers or nurses, especially those who have had a smattering of dietetics, give their babies a wide and studied variety of food, these will often make known decided preferences for only certain articles of diet and will not eat others. Habits now formed often last through life and we have that embarrassing and stupid person who simply cannot eat everything that is set before him as a normal, proper person should. The intelligent mother must be helped to choose that intermediate course that is best suited to her particular baby. I am firmly convinced that these things are of great importance, not only in forming proper habits of eating, but also in the formation of that bigger thing we call character.

I have purposely omitted the discussion of eggs and of beef juice in the dietary of the first year. Eggs have enjoyed great popularity until recent times. So much has been said against them in recent years that one would almost expect to find them classified among the dangerous articles of diet. It is rather difficult to explain or to understand or to share in this change of view. That they are a wholly unnecessary article of diet is certain. That they are demonstrably objectionable, except in certain dyscrasias, or in those rare cases where idiosyncrasies are present, has never been shown. That the ingestion of egg occasionally gives rise to anaphylactic reactions such as urticaria, egg asthma, etc., is well known but this does not affect its status as food except in those rare individuals. Mothers should always be warned when giving egg for the first time, no matter what the age of the child, that only the smallest amount should be given the first few times until absence of idiosyncrasy is established. As to beef juice, I have never been able to satisfy myself that it offers anything tangible, or demonstrable, that is not found to an equal extent in broth, and it is far more expensive and wasteful. While both the protein and the extractives are 2 or 3 times as high in beef juice as in broth one commonly gives so much more of the latter at one time that these differences are equalized.

How Long Shall the Bottles be Given?—In nearly all of our textbooks, in the popular books and articles for mothers, and in the Government Manual the advice is given to wean the baby from the bottle at about the end of the first year or shortly after the beginning of the second. My own experience has never lead me to be reconciled to this advice. It matters little how a baby takes his milk after the first year but there is practical unanimity that has been greatly strengthened by recent investigations that he should commonly have nearer a quart than a pint of milk a day during the second year. The baby that will spontaneously quit his bottle under intelligent care is very rare, and so long as he takes a bottle one is very sure that he will take as much milk as we want him to. While some babies will take as much milk from a cup as from a bottle at this period when discontinuing of the bottle is advised, it is my experience that they are in the vast minority. It is certainly the commonest experience that the baby that is weaned from the bottle at this time will not take nearly that amount and not infrequently soon refuses milk altogether. The only possible objections to continuing the bottle longer would seem to me to have to do again with feeding psychology, and with the trouble of preparing the bottles. While the latter is commonly mentioned by mothers it becomes negligible if the matter is explained. As to feeding psychology—it is true that a baby will often take a bottle indefinitely as is attested by the eagerness with which older brothers and sisters like to "finish the baby's bottle," and by at least one case that I know of in which a young lady of 18 years simply could not take milk except from a bottle. As a matter of fact it is very easy to wean a child of three or four from the bottle and if he will then refuse to take milk from a cup it is not of as vital importance as it is earlier. It is my own practice to advise giving one or two feedings from a cup shortly after the first year, but I would allow, or rather recommend, the continuation of the early morning, the bed time, and the pre-nap bottle into the third or even the fourth year. In this way they are assured at least two or three 8 oz. bottles a day and are acquiring at the same time the cup habit. If the baby will take the milk early and adequately from a cup, as most of them do *not* in my experience, then there is, of course, no objection to that method alone.

Feeding during the Second Year.—During the early part of the second year four feedings a day are ample, later in the year, say the fifteenth month, three are enough, with the addition of the juice of an orange given once a day between two feedings. Milk is given at each meal not to exceed a quart a day, often a less amount is preferable. The manner of giving this has been discussed in the preceding paragraph. The general arrangement of meals and the kind of food at each meal is much the same as in the latter part of the first year. The details can be left to the mother or nurse provided always the child gets a well balanced daily ration of milk, cereals and puddings, at least one vegetable, potatoes or rice, fruit juice or fruits that are permitted, broth or beef juice, and in the latter part of the year meat. Vegetables such as turnips, asparagus, squash, stewed celery, peas and lima beans can gradually be added.

At about the middle of the second year meat may be added once a day, beginning at first with scraped beef, to be followed soon by finely cut up rare roast beef, or steak, white fish, chicken, or turkey. If desirable this can be alternated with an egg to which there is no special objection. Scraped beef is made from round steak either by searing both sides in a hot skillet and scraping with a knife or spoon; or else by grinding it fine in a meat grinder *at home* and lightly broiling it in the form of a cake or patty. A tablespoonful of this can be given daily at the eighteenth month, or even at the end of the first year if it seems indicated because of anemia, rickets, or some peculiar digestive condition. There is again no great advantage in variety of meats and the heart of a lamb chop lends itself well to daily use after the scraped beef period. Bacon is commonly taken eagerly and may be allowed daily. Among the articles to be especially avoided are such things as grapes, unscraped apple, corn, and unripe and not finely mashed bananas, radishes, cucumbers, tomatoes, pickles, pears, or peaches. All fruits and vegetables, because they do not soften in the stomach, and are not yet properly chewed, must be given finely mashed or divided. Honey, jellies, gelatine, and lighter preserves and marmalade are freely permissible during the second year.

The question of whether *the heavy meal can be given in the evening* always comes up during the second year especially if the child has taken his place at the family table. It has always seemed to me that the heavier evening meal has been condemned more than seems warranted by the facts. Theoretically, it is hard to see why meat, potatoes, and vegetables if properly prepared for the child should be any greater factor in causing a restless night than is milk, and my own experience would lead me to conclude that there is no objection to the heavier evening meal during the second year, and certainly not after that time.

Feeding after the Second Year.—The three meals are naturally continued and it is still desirable to give at least a pint of milk a day. Commonly more milk is preferable, but never over a quart, unless it interferes too much as it sometimes does with the taking of other food. If milk is refused good butter should be given freely. Milk can often be given more easily in the form of weak cocoa and to this there can be no objection. After the second year the question commonly is not so much what to give as *what not to give* to a child provided always the diet is well balanced as indicated above. The subject may be considered under the following heads:

Fruit.—While many of the fruits are permissible if ripe and used in moderation, they are a source of distinct danger if these requirements are not met. Thus grapes, bananas, peaches, pears, apples, plums, berries and even oranges can if eaten in excess, or if not ripe, cause most violent gastro-intestinal symptoms, vomiting, diarrhea, pyrexia, and even convulsions. It is, therefore, well to be cautious about them all through childhood and especially up to the fourth year. Bananas are commonly placed wholly outside the pale, and hardly on sufficient evidence. A well ripened banana with the peel turned nearly or completely brown, or at least a deep yellow with brown spots, that is soft and mealy is an excellent article of diet that can be given even in the second year or earlier if properly mashed. An unripe, pale yellow, or green tinged banana should not be given to any child of any age. Baking or broiling makes them more digestible. Peaches, pears, and apples can be given in moderation if well chewed, or mashed, or *scraped*, in the third year, even in the second year, if thoroughly ripe—if not thoroughly ripe or if swallowed in large pieces they should be avoided all through early childhood. The same is true of such berries as strawberries, raspberries, and blackberries. All of these foods can be given cooked earlier than raw, and in the case of berries especially this has also a bacteriological value as they cannot be thoroughly cleansed.

Vegetables.—All such vegetables as corn, cucumbers, cabbage, onions, radishes, lettuce, raw celery, beets, and eggplant are best avoided wholly during the first three years and some of them even for a much longer time. Corn, cucumbers, and especially radishes are peculiarly indigestible and if taken freely can lead to the same violent symptoms mentioned under fruits. Ripe tomatoes can be allowed earlier than the others, and when stewed and finely divided can be given in the third and even the second year.

Meat.—Certain meats that are peculiarly heavy in fats such as pork other than bacon; goose, duck, and especially all kinds of sausages should be used in great moderation if at all even up to the sixth or seventh year. Other meats to be used with equal caution are salt fish, game, kidney, and liver.

Bread, Pastry, and Desserts.—Hot fresh breads and rolls are always more indigestible than stale, dried or toasted breads, zwiebach or crackers, probably largely for purely mechanical reasons, and should be avoided in the first three years. Griddle cakes and waffles should be used very cautiously before the fourth year both as to quality and quantity. All lighter puddings such as bread, tapioca, rice, and arrowroot puddings, custards and blanc mange are permitted freely in the second year and even in the latter part of the first year, but plum, raisin, date and other heavy puddings should be avoided in early childhood. The same applies to pies and the heavier sweeter cakes. Cookies and sponge cake can be allowed in the second year with meals. Plain pure ice cream in connection with meals seems harmless after the second year.

Candy.—The question of candy is not simply one of digestibility. Simple, pure candy made up of sugar, chocolate, and harmless flavoring agents can hardly be looked upon as harmful, *per se*, if used in moderation. This is especially true if it is treated as a sort of dessert to be given only after meals. Unfortunately the taste for such sweets once aroused commonly leads to a demand for them in season and out of season. The child commonly learns the degree of teasing necessary to break down the barriers of resistance that are only too often not very strong. Temporary peace is generally bought at the cost of discipline, and the effects of this are far reaching. Good pure candy is harmless only when there is unquestionable discipline. Even then it should be permitted only *after meals* because it seriously interferes with the normal appetite for more desirable food if given between meals. Heavy nut candies and excessively sweet bon-bons should, of course, never be given during early childhood. Inasmuch as candy is never given because of its food value, but primarily because of the pleasure it gives the donor in seeing a child made happy; and its use is accompanied by such serious disciplinary and even nutritional harm; it is well to omit it entirely during the first few years of life, and emphatically so in unseasonable times and in excessive amounts.

Nuts are a wholly unnecessary article of diet and are quite indigestible. They should, therefore, be omitted entirely during the first two years of life, and allowed only with caution after that time.

Drinks.—Tea, coffee, and all alcoholic beverages have, of course, no place in the dietary of childhood. What has been said about candy applies to ice cream sodas, cones, etc.

THE USE OF OTHER MILK THAN THAT OF THE COW IN ARTIFICIAL FEEDING

From purely local geographical reasons on the one hand, and from therapeutic reasons on the other, the milk of other mammals has been used from most ancient times down to the present time. While that of the goat and the ass have been used chiefly for therapeutic purposes, that of the goat, the reindeer, the llama, the camel, the water buffalo and others have been used in various countries where these animals are the predominating, or only, milk givers. Two methods have been in vogue, both therapeutically and for local reasons, during all historical times, that of direct nursing from the animal and that of indirect feeding with the milk after it is drawn.

Direct Nursing from the Udder.—Among the ancients direct nursing was practiced not only among mortals, but legendary heroes, too, and even the gods were pictured as having taken their early nourishment and often their attributes from some animal or other directly.[2] From the Greeks we hear that Telephos, the son of Hercules, was nursed by a doe; Pelios, the son of Neptune, by a mare; Aegystus, Dionysius, and Æsculapius, each by a goat; Orion by a bear; and Cyrus by a bitch. Even Zeus himself was said to have been nursed by the goat Amalthea on the Island of Crete. The Romans pictured their legendary founders, Romulus and Remus, nursing a she-wolf. In Romance too the same theme occurs again and again. Daphnis was nursed by a goat, Chloe by a sheep, and in our own times Kipling's Mowgli was reared by a she-wolf.

Throughout historical times direct nursing is recorded for many centuries.[2] Among the ancient Egyptians direct nursing at the goat or at the cow seems to have been practiced to a considerable extent. The same is true of the Scythians and other peoples. During the eighteenth century nursing from the goat and the ass was used to some extent in France as a therapeutic measure. From the year 1816 an interesting book has come down to us on "The Goat as the best and safest wet nurse (Säugeamme) by Zwierlein of Bad Bruckenaw."[2] By this time syphilis had laid its restraining hand on the wet nurse problem and for this and other reasons Zwierlein enthusiastically advocated direct nursing of the goat as the best substitute for mother's milk and a more natural one than the boiled cow's milk that was then used to some extent. In this and two subsequent publications he gave minutest directions as to the choice of a goat, the position in which to be nursed, with elaborate details as to restraining devices, benches, etc. together with strict directions as to nursing hygiene. The baby was to be nursed every two or three hours at first, but after the sixth week only four times a day. Direct nursing became for a time very common. The excellent results reported by Zwierlein can, of course, be accounted for only by contrasting them with the gruesome results of the bottle feeding of the time. As might be expected the bad results soon became evident to those less enthusiastic than Zwierlein and direct nursing fell into abeyance.

It was again revived in the latter part of the nineteenth century in France by Tarnier, Fournier, Parrot, and others.[2] Tarnier at first used the goat but with such poor results that after 1881 only asses were used. With these the results were so good that the bottle was completely abolished at the Maternité, in Paris. Parrot adopted the same measure in 1881 and 1882 for syphilitic infants. These could not be put directly to a wet nurse, and he hoped also to cure them by giving mercury and iodides to the nursing asses, with such results as one might imagine. Parrot reported among those fed on cow's milk a mortality of 83 per cent.; among those nursed directly from the goat 67 per cent.;

and those from the ass only 21 per cent. Elaborate arrangements were made in the *Pavillion Parrot* for this method of feeding, the asses' stable being placed close to the nursery with a covered way between. After Parrot's death the practice gradually fell into disuse, partly because of that fact and partly because of the cumbersomeness and great expense. The yield of only about one quart a day from each ass made it cost over 2000 francs a year to feed one baby.[2]

Sporadic attempts were made for some years to renew the practice of direct nursing both in France and Germany, but in recent years with better results from feeding modified cow's milk it no longer seems likely that the attempt will again be made in a medical way, unless as Marfan has suggested a goat can gradually be developed that will have a more human like milk. There is furthermore no evidence of any kind that direct nursing has any advantages over feeding with milk that has been procured and kept under sanitary conditions. At the present time direct nursing is practically restricted to the use of the goat in such countries as Cuba, Mexico, Turkey, and certain tropical countries.

Indirect Feeding of Other than Cow's Milk.—Only the milk of the *goat* and the *ass* are of sufficient importance to warrant discussion. The following table, compiled from various sources, gives at a glance the approximate comparative composition of human, cow's, goat's, and asses' milk.

	Human milk	Cow's milk	Goat's milk	Asses' milk
Protein	1.50	3.50	3.50	2.00
Fat	4.00	4.00	4.00+	1.00
Milk sugar	7.00	4.50	4.50	6.00
Salts	0.20	0.75	0.90	0.45

Goat's milk is used extensively in Switzerland, Germany, Cuba, Mexico, and other countries, and to some extent among the poor, especially the Irish, in our own larger cities. The goat has been called the "poor man's cow" because the initial cost is small, she takes up little room, produces twice as much milk per body-weight as the cow, actually yielding 10 to 12 times her weight of milk annually, and while she eats more in proportion to her weight than the cow, she has a notoriously wide range of food that is relatively cheaper.

Goat's milk has been tried extensively in infant hospitals and elsewhere as a substitute for cow's milk, even in most recent times, at first undiluted, later modified just as cow's milk is modified. The composition and behavior of the two milks is practically identical so far as is known. The casein is generally said to coagulate into more tough and compact masses than does that of cow's milk, though the evidence is not conclusive and may be largely hearsay. The fat globules are smaller than those of cow's milk and of more uniform size. On standing practically no cream layer is formed and the cream cannot be separated by the centrifuge.[20] Much has been made of the value of this latter point in infant feeding in the extensive propaganda that has recently been made to popularize goat's milk as an infant food. That goat's milk has any superiority as an infant food, either in the fresh or in the canned or evaporated form, as compared with cow's milk, remains to be demonstrated and I think in our present state of knowledge can be said to be doubtful. The greater freshness in localities where fresh cow's milk cannot be procured is of some advantage. Goats, too, are said to be relatively immune to tuberculosis, possibly due rather to less insanitary herding such as cows are commonly subjected to, than to an innately greater immunity. Much has been said about the superiority of certain races of goats over others though the evidence here again is not clear and at the present time the subject has little of practical interest.

Goat's milk has a peculiar odor and taste that can perhaps best be described as "goaty." This is perhaps in part due to pollution with intestinal excreta[20] but more probably to a specific acid secretion of the skin.[14] While this might be objectionable to the older child or adult it would have no influence on the baby.

Asses' Milk.—The case for the use of the milk of an ass as a substitute for cow's milk in infant feeding, *and that in its undiluted form*, is a much stronger one. Asses' milk has been used extensively for feeding babies in Spain, Italy and Southern France. In England, too, Cheedle tells us as late as 1902 that "in London there are regular establishments for the supply of asses' milk."[35] In this country my friend Dr. Harrell of San Antonio, Texas, tells me he has used quite extensively in the feeding of babies for whom one would commonly like a wet nurse, the milk of the indigenous burrow with very gratifying results. From all sources the reports have been uniformly favorable to the use of asses' milk for this type of case. The good clinical results would be anticipated from the chemical composition. The protein is practically identical in amount with that of human milk, both as to total quantity and as to the relative proportion of casein and lactalbumen (1 to 1); the sugar is of approximately the same amount; the fat is very low, approximately 1 per cent.; and the salts approach in amount much nearer to those of human milk than do those of cow's or goat's milk. It sours less easily and coagulates, as we might expect, very slowly and imperfectly in small flakes. When boiled it forms a flocculent coagulum, due to the large amount of albumen, which is, however, so fine that it passes through the nipple. The fat globules too are said to be very fine. The small amount of fat might make the milk unsuitable for prolonged use but this would be no serious objection as the use of this milk is nearly always temporary.

Unfortunately asses are not available with us for practical use. They are furthermore relatively expensive because of the small individual yield of milk. The ass is not adapted by years of domestic development for milking purposes as is the cow, and requires the stimulus of the sucking colt to keep up the milk supply. As a result a quart of milk is about the average practical daily amount that can be procured. If asses' milk could be obtained in quantity like cow's milk there is every reason to believe it would be of greater practical value in infant feeding, especially in infant hospitals. The whole thing cannot yet be dismissed and it may again crop out in more practical form. For the present so long as asses' milk is more difficult to obtain and more expensive than wet nurse's milk it is not likely to come into favor.

What has been said of asses' milk is probably largely applicable to *mare's milk* though adequate clinical and experimental data are wanting.

PROPRIETARY INFANT FOODS. "BABY FOODS"

If the proprietary or patented "baby foods" were not so widely used they would hardly merit discussion in a scientific consideration of infant feeding. The fact that probably the great majority of babies in this country are fed one or the other of these preparations would seem to make it incumbent to discuss them here.

The wide spread use of these so-called "foods" is mainly due to the following reasons.

1. They have been so widely and skillfully advertised to both laity and physicians as infant "foods," that they have come to represent to a very large proportion of both laymen and medical men the *alpha* and *omega* of infant feeding. The very word "food" carries with it the idea of finality, of a system of feeding in contrast to the systems represented by other "foods." The process of feeding babies consists thus viewed of trying to find the "food" that fits the baby in question. Physicians are swamped with mail, and detail men each pointing out the peculiar virtues of the particular food represented, and at the same time presenting them with a simple system of feeding this "food." The mothers too are assailed with alluring literature and in a few cases are given long distance help from the "home office," and by local nurses, in their feeding problems. One of them at least has extensive methods of finding out the names of parents of newborn patients, or of those who for the time are using their food, and sending to them encouraging literature with the offer of help by correspondence.

2. They appeal to the unknowing mother, allured by the claims of the manufacturers and by the recommendations of neighbors and friends, as a complete method of feeding the baby without the necessity, bother, or expense of seeing a doctor.

3. They appeal furthermore, for reasons already discussed, to a large number of physicians, who consider the infant feeding problem too difficult, or too insignificant, to merit a mastery of it on their part. The "baby food" offers a ready solution with its simple directions easily mastered, and if the favorite one does not seem to fit there are others to try. Often the system need not be learned, the mere name is enough and the mother herself can follow the directions that accompany the "food."

4. Some of these preparations, *i.e.*, those used without fresh milk, are peculiarly easy to digest and lead to comfort, gain in weight, and apparent well being on the part of the baby. If the mother is warned that her baby is merely fat, not necessarily healthy, that it is very likely to get rickets and possibly even scurvy and has little resistance and immunity to disease, she has only to see how fine her baby is really getting on and easily attributes the warning, with a knowing smile, to the "peculiar prejudice doctor's have to baby foods!" As one mother said when warned not to continue a certain food too long "but the company wrote me and said it was a complete food in itself and much safer than cow's milk."

For *practical purposes "infant foods" are divided into two classes:*

1. Those that contain milk and are advertised as complete foods in themselves.

2. Those that are to be used only with fresh cow's milk and are so advertised.

It would manifestly be impossible and unjust to make the same blanket criticism of all of them when they offer such wide variations in composition and mode of use. Only those more widely used in this country will be considered. The comparative composition of some of the different preparations can be seen at a glance in the following table taken chiefly from Morse and Talbot.[23]

In the first class as the chief representatives are condensed milk, malted milk, and Nestle's Food. Condensed milk is commonly milk evaporated to about one-fourth of its volume, to which is added over 40 per cent. of cane sugar. Unsweetened condensed milk, commonly called *evaporated milk*, and *dried milk* such as Dryco Milk or Klim are simply milk or partially skimmed milk condensed or dried by various methods, without the addition of further carbohydrates, and do not enter into the present discussion. Condensed milk, i.e., the sweetened preparation, is probably used more extensively than any other "infant food." In malted milk (Horlick's, Borden's) and in Nestle's Food the milk is evaporated to a powdered form and sugar and partially or completely dextrinized flours are added. In malted milk the additional carbohydrate is composed of dextrins and maltose. In Nestle's Food the additional carbohydrate is made up of about 50 per cent. of nearly equal parts of dextrins and maltose, and of cane sugar, together with about 15 per cent. of unconverted starch. It is evident from a glance at the table that all of these foods contain well over 50 per cent. of sugar, with from 5 to 10 per cent. only of fat, and from 8 to 16 per cent. of protein. A simple comparison of all of them with the composition of mother's milk shows how widely they differ from the infant's natural food. The fat is strikingly deficient, in some as low as one-tenth of the sugar. The sugar is depended upon in all of these "foods" because of its physiological kinship to fat to make up for the deficiency of the latter. There is every reason to believe that such extensive substitution is attended by serious nutritional risk. The absence of fresh animal proteins is another obvious and serious objec-

Food	Percentage composition					
	Fat	Protein	Sugar		Starch	Ash
Condensed Milk Eagle Brand	9.61	8.01	54.94	Cane 42.91 Milk 12.03	0	1.78
Horlick's Malted Milk	8.78	16.35	67.95	Milk, Malt 49.15 Dextrins 18.80	0	3.86
Nestles Food	5.50	14.34	58.93	Milk 6.57 Cane 25.00 Malt, Dextrins 27.36	15.39	2.03
Mammala	12.12	24.35	55.34	Milk	0	4.93
Mellin's Food	0.16	10.35	79.57	Malt 58.88 Dextrins 20.69	0	4.30
Soxhlet's Nährzucker*	0.03	0.13	95.51	Malt 41.16 Dextrins 54.35	0	1.70 (0.84NaCl)
Mead's Dextri-maltose[1]	0	0	93.00	Malt 52.00 Dextrins 41.00		2.00 (1.80 NaCl)
Mead's Dextri-maltose[3]	0	0	93.00	Malt 52.00 Dextrins 41.00		2.0 (2.Pot. Carb.)
Eskay's Food	3.52	6.70	55.82	Milk 54.12 Dextrins 1.70	29.90	0.99
Dennos' Food*	1.17	11.10	16.64	Cane 13.32 Dextrins, etc. 3.32	57.00 Cellulose 4.27	0.79
Imperial Granum	1.04	14.00	1.80	Dextrose 0.42 Dextrins 1.38	73.54	0.39

*Taken from Mellin's Food Company Analysis.

tion. For reasons that have been detailed sugar is well borne in such a medium that contains a minimum of fat and that possibly influenced by the process of preparation; and a protein that is not only very small in amount but in such a state that coagulation is almost imperceptible in the stomach.[7] For these reasons these foods are peculiarly easy to digest and the babies become fat and comfortable, at times even firm with a sort of pale rosiness, but commonly they are ultimately pale, flabby, inactive, and often seriously rachitic. It is these foods largely that kept the problem of scurvy alive. The greatest danger in the *temporary* use of these foods lies in the fact that the mother is apt to make them permanent. The very fact that many babies even after living on these foods for months *look* well-nourished to the superficial observer would only emphasize this insidious danger that lies in their use. That these foods, however, and especially condensed milk, are well nigh specific in the treatment of just those conditions in which fresh cow's milk does not lead to results gives to them a distinct field of usefulness under certain circumstances where more complex foods are not feasible. The fact that the mother may act unwisely in the future in spite of adequate warning would hardly deter one from giving the baby the therapeutic benefit of one of these foods if it seems indicated and if under the given circumstances no other food of equal therapeutic effect can be prepared or procured.

In the second class belong:

(*a*) *Malted foods*, such as Mellin's, and Horlick's Food that are composed largely of dextrins and maltose, with the latter in decided excess especially in Mellin's; and Soxlet's Nährzucker and Mead's Dextri-Maltose in which the two carbohydrates are more nearly equally represented, the maltose being even less than the dextrins in the Soxlet preparation. The former contain an appreciable amount of proteins due to the fact that they are made by the diastasic action of malt on wheat and barley; while the two latter contain almost no protein probably because made from more nearly pure potato or corn starch by a different method. This difference offers no appreciable therapeutic advantage. Mellin's contains about 2.5 per cent. bicarbonate of potassium. Mead's Dextri-Maltose is not advertised to the laity and does not properly belong in this present discussion. For this reason it is preferred, as it is obviously desirable, to avoid that factor. It is put out in three forms: Dextri-Maltose No. 1 containing 2 per cent. of sodium chloride; No. 3 containing 2 per cent. of potassium carbonate; and No. 2 containing neither of these.

All of these preparations are used only with fresh cow's milk and differ from one another practically only in the relative amounts of the two carbohydrates and in the presence or absence of the potassium and sodium salts. They meet the very extensive present demand for a dextrin-maltose preparation as a substitute for milk and cane sugar. That they have peculiar virtues as "milk modifiers" other than that which resides in the added potassium salts is not evident clinically.

(*b*) *Farinaceous, or cereal, or starchy foods* such as Imperial Granum, Ridge's Food, Robinson's, Johnson's and the other barley flours. The barley flours are commonly used in the preparation of barley water. Imperial Granum is a cereal food that can be added to the bottle in the pre-spoon period. That it has any advantage over flour ball, or simple wheat or barley flour, used in the same manner, is not evident. When given in the first few months as directed it can easily lead to starch indigestion.

(*c*) *Foods that contain both sugar and starch*, of which the two chief representatives are Eskay's Food and Dennos' Food. The former is composed of 50 per cent. of milksugar with about 30 per cent. of starch, with the addition of egg albumen, the advantage of which seems apparent only to the manufacturers. The latter contains about 60 per cent. of insoluble carbohydrates (starch and cellulose) and about 13 per cent. of cane sugar. The objections to "baby foods" becomes in a way most apparent in such preparations as these that are widely advertised and used. Manifestly they offer no advantage to the intelligent physician over the use of simple milk or cane sugar, together with a starchy food, such as are used in a simple milk and added carbohydrate mixture; they admit only of a clumsy parallel flexibility of the different carbohydrates by increasing or diminishing *all* of them, instead of using them separately as seems indicated; and they carry with them all of the objections that apply to this class of patented foods.

The chief objections to the "Baby Foods" as a class are the following:

1. They are unnecessary.
2. They are expensive except in a few cases.
3. The use of the word "food" that leads the uninformed to think of it as the important thing, and not the milk that is the real basis.
4. They offer a system of infant feeding that eliminates scientific individualization, the keynote to successful feeding.
5. They are patented and advertised to the laity, often directly to the mother, and in a few cases by devious paths. *They leave the accurate diagnosis and the indication for individual treatment of one of the most complex and fateful problems in medicine to the mother and the directions on a tin can or a glass bottle.* The physician who contents himself with recommending these preparations to be used "according to directions" becomes *particeps criminis* in this illogical and dangerous business.

It is often hard to know where to draw the line. The physician who would scorn to use any patented baby food with its ready made system of feeding may be somewhat confused when he finds that the can of milksugar and the box of barley flour that we all use also offers to the mother or the physician a system of infant feeding with milksugar or barley flour. Ultimately the conscientious practical physician will use one or the other of these preparations if he sees an indication for its use that is not met in that particular case in its particular environment by some other more regular preparation. He may prefer at one time a dextrin-maltose preparation with a high maltose content such as Mellin's Food because of its laxative action; another time one with a higher dextrin content such as Soxlet's Nährzucker when there is a tendency to looseness of the bowels; or he may wish to avail himself as a temporary expedient of the unquestionable therapeutic action of the condensed or evaporated milk group. He will, however, use them only after due explanation and serious admonition to the mother and never "according to directions."

GROWTH AND DEVELOPMENT OF THE ARTIFICIALLY FED BABY

Under favorable circumstances, artificially fed babies can be brought up so that they seem little, if any, inferior to the breast fed baby, even if they have never had any breast milk. While there is no reliable tangible evidence that such babies are handicapped later by remote unfavorable effects, such, for example, as less vigor, or a diminished resistance to disease, one always feels that that possibility should be removed whenever possible, by giving every baby some mother's milk for a time. If the baby can have breast milk for only a few months, or even a few weeks, the problem of artificial feeding becomes greatly simplified, the results surer. In general, one may say that the artificially fed baby does not progress with that well-nigh absolute assurance that the breast fed baby does; that its progress is less rapid and less perfect, as a rule, and that minor, as well as the more serious, digestive and intestinal disturbances are far more frequent. Under favorable circumstances, perhaps 50 per cent. can hardly be distinguished from the breast fed baby; another 25 per cent. look less rosy, are thinner, are less comfortable, and have a tendency to minor disorders of digestion; another 15 or 20 per cent. give considerable concern, rarely as to life, however; while in perhaps 5 per cent., serious difficulties are encountered throughout the first four or five months. The younger the baby, the more difficult the feeding. The second half year is nearly free from troubles, and the "second summer" is now encountered only as a tradition handed down by those whose memories date back a generation or two. While in general, the gain in weight is less than during the first few months, and the baby is less rosy and comfortable than the breast fed baby, in the second half year the gain is commonly more rapid than in the latter, and by the end of the year they are alike in weight and seem nearly equally well nourished.

Of the greatest importance is the avoidance and immediate rectification of all digestive disorders, even the most trivial. In the breast fed baby, these tend to spontaneous recovery; in the bottle fed baby they are the forerunner of the more serious disorders. For this reason the presence, or absence, of immediate and unqualified coöperation on the part of the mother or nurse is commonly the factor that decides whether progress shall be normal, or halting. For this reason, too, in the first few months one must be satisfied with such a gain in weight and such an amount of food only as will insure the avoidance of any digestive disturbances. While in many cases this means relatively slow progress during the first four or five months, in some few cases, even candid explanation to the mother that there will be very little gain in weight, a rapid and favorable progress can be promised as soon as the baby is put on cereals, broth, orange juice and vegetables, in addition to the bottle.

Normal Feces of the Artificially Fed Baby

The breast fed baby has a normal standard stool to which all breast fed stools can be compared. The artificially fed baby has no single standard stool. The normal stool here depends upon what food combination the baby is getting. The soft, mucous, greenish-brown barley water stool is just as normal as the formed pale yellow, offensive stool of the modified cow's milk fed baby, or the rich brown pasty stool of the Nestle's Food baby. It cannot be emphasized too strongly, for such emphasis still seems necessary, that the soft, mushy, gold-yellow, slightly acid, aromatic, non-offensive stool of the breast fed baby does not form our standard of what the stool of an artificially fed baby should be like. The cow's milk fed baby's stool that even remotely resembles that of the breast fed baby should be considered pathological and demands a change in food. The breast fed baby that gets enough to eat nearly always has two to three, or even four or five, stools a day *that are never formed;* the bottle fed baby has usually one, or at most two stools a day, and then often only with assistance, and they are formed as in the adult, or of the consistency of a heavy paste. They are, furthermore, of a pale yellow color, alkaline in reaction, and with a stronger, more offensive odor of decomposition, not of fermentation, due to the larger amount of protein. The uniformed mother who interprets the baby's stool, in the light of her own troubles, is apt to be contented if the baby only has "plenty of movements;" the experienced physician considers even one soft stool too many, as a warning to be heeded, and knows that a formed stool is normal or can easily be remedied without detriment to the child.

Since the nature of the artificially fed baby's feces varies with its multiform food, it is evidently of the utmost importance that every physician who feeds babies should familiarize himself practically, by routine examination, with the gross appearance, color, odor and consistency of the stools that normally follow the use of different mixtures. Only in this way can he recognize the abnormal. *This examination must at all times and in every case be made personally.* It is a very familiar fact that a description of the stool by the mother, or even by the nurse, is not only inadequate, but often wholly misleading. Successful feeding requires the immediate knowledge and rectification of the least deviation from the normal, for at this stage the remedy is simple and effective, and the more serious disorder is obviated.

The factors that influence the characteristics of the normal stool can be considered under the following heads:

The fat, in general, tends to give the stool a pale yellow color. If in excess, and no other disturbance exists, the stool becomes very pale, gray or whitish, and either soft and greasy, or dry, hard and foul. The fat in certain combinations acts as a laxative; in others, it is very constipating. In mixtures low in protein and high in sugar, the former is apt to be the rule; if high in protein and low in sugar, the latter is the more normal reaction.

The Protein.—If the fat is practically eliminated from the food, as in the use of skimmed milk or fat free buttermilk, the effect of the protein on the normal stool becomes evident. The stool becomes pale brownish in color, has a strong, cheesy or foul odor, a strongly alkaline reaction and a peculiar smooth vaseline-like consistency and appearance that is *sui generis.* If fewer and more constipated, they are darker brown and dryer; if more frequent, they are pale grayish-brown, or brownish-gray and moister. In general, the protein is constipating unless in great excess, when it can lead to diarrhea and to such stools as have just been described. The constipating effect of the proteins is sometimes used therapeutically by increasing the amount of casein at the expense of the sugar, as in Eiweiss Milch, or by adding dried, powdered casein, such as plasmon, sanatogen, or pure dried casein, to the mixture. The protein probably acts here by partially substituting a decomposition for a fermentation process.

The *carbohydrates* give the stool a browner color, and with their normal fermentative action, tend to make it softer, more frequent, more acid and less offensive; while in pathological conditions, milk-sugar and cane sugar seem more laxative than dextrin-maltose preparations, the opposite seems true in the baby with a normal stool. This requires the further statement that only those dextrin-maltose combinations in which the maltose greatly exceeds the dextrins, as in Mellin's Food, are distinctly laxative; while those like Soxlets' Nähr zucker, in which the dextrins materially exceed the maltose, are relatively constipating. The dextrin-maltose preparations, especially Nestle's Food and Mellin's Food, give the stools a richer, deeper brown color than do the other sugars or the starches.

The *starches* have the same general effect on the stools as the sugars. In normal conditions when they are well tolerated they have, however, a constipating effect as compared with the sugars. The *dextrins* seem to occupy an intermediate position in this respect. If the starch is not tolerated, or is given in excess of toleration, it undergoes fermentation and leads to diarrhea. Starch in the form of cereal waters, *if tolerated*, has an indirect constipating effect also because of its effect on the casein curd, rendering it smaller and more readily digestible.

Animal foods, such as broth, beef juice, etc., cause the normal stool of infancy to approach that of the adult in color, odor, reaction and consistency.

The *salts* have a marked effect on the normal stool. The effect of those innate in the food itself is not clearly defined. The addition of salts, like *sodium citrate* and *sodium bicarbonate* and of alkalies like *lime-water* and *milk of magnesia* (magnesium hydroxide), tends to constipation, probably because of the effect on the casein curds. Magnesia has, however, a laxative effect in itself that much more than balances its constipating effect as an alkali. The *potassium salts* that are added to such dextrin-maltose preparations as Mellin's Food and Mead's Dextrin-Maltose No. 3 are claimed to have a laxative effect. They are added, on the other hand, to Malt Extracts to make them less laxative by counteracting their acidity.

The starvation or hunger stool must be classified with the normal stools if it is a result of therapeutic withdrawal of food; with the abnormal, if it is the result of pathological conditions that lead to starvation, such as vomiting, pyloric or esophageal stenosis, and like conditions. If a baby gets no food at all, and still has movements, as it commonly does, they are ordinarily *small, always loose or semi-liquid, more or less transparent and mucus-like, of a dark greenish-brown color, and have a faint, old, stale, musty, non-fermented and non-decomposed odor.* Such a stool is composed only of intestinal and glandular secretions, chiefly mucus, bile and bacteria. If the baby is on *barley water* or some other thin carbohydrate mixture, it will have normally such a *hunger stool,* because the carbohydrate is completely absorbed and no food remnants appear in the stools. If the carbohydrate is in excess of tolerance and undergoes bacterial fermentation, the hunger stool is replaced by one very similar in appearance, except that it is frothy and acid. As soon as milk is given to the starving baby the stool becomes more normally yellow and offensive, and if well digested, firmer in consistency. *One of the commonest and greatest mistakes in infant feeding is to interpret the perfectly normal hunger stool of the baby on a barley water diet as abnormal, because the stool is "greenish, liquid and slimy."* Babies are often kept, to their invariable detriment, for many days on barley water, simply because the stool does not become yellow and firmer. A yellow firm stool on a barley water diet alone would be farther from normal than a greenish watery stool on a milk diet, for its occurrence is impossible.

The *cathartic stool* deserves a separate discussion that it has never received. It is best considered under the discussion of the *normal stool,* because it is a normal reaction to a therapeutic measure. If a mild cathartic is given the baby the only apparent microscopic change in the stool is that it becomes softer, moister, and for a time more frequent, depending on the cathartic. Depending again upon the cathartic, this is due in different degrees to an increase in the intestinal secretions, and to an increase in peristalsis which sweeps out the liquid intestinal contents before they have time to lose part of their moisture in the lower portion of the large bowel. If a large dose is given, or a rather drastic cathartic, the stool becomes liquid, and after all the undigested remnants are expelled, full of mucus and of a greenish-brown color obviously identical with the hunger stool. Such a stool is as manifestly normal, under the circumstances, as it would be abnormal if no cathartic had been given. This would hardly seem to deserve mention were it not for the fact, as I have pointed out elsewhere,[36] that the cathartic stool, exactly like the barley water or hunger stool, is so commonly interpreted as abnormal and calling for further *therapeutic starvation* and *cartharsis.* One of the commonest things in practice is to see the baby with a normal hunger-cathartic stool, *because of that stool* kept on a further starvation or barley water diet for days, with the addition of a daily dose of castor oil or calomel, in the vain anticipation of the yellow, firmer stool that will never come till the crack of doom unless milk is added to the food. *The starvation stool and the cathartic stool call for food, not for further starvation and catharsis.* In itself, starvation is always harmful, though it may be necessary for a brief time, to correct a greater evil. *That it is never necessary to starve a baby more* *than 24 hours, or to give more than one initial dose of castor oil, would seem axiomatic rather than disputable.*

ABNORMAL FECES OF THE ARTIFICIALLY FED INFANT

Abnormal Density.—*The constipated, pasty, or formed stool* is normal in the artificially fed baby unless it is extremely hard and dry. Even though the condition is severe enough that the baby in spite of dietetic measures cannot pass it, and mild cathartics, such as milk of magnesia, are necessary, that is not abnormal enough to cause concern or to be harmful to the baby now or hereafter. The dry, hard, gray, foul soap stool that occurs when a baby gets an excessive amount of milk, that does not lead to an acute digestive disturbance, is not only abnormal and harmful in itself, but is commonly accompanied by a serious metabolic disturbance that requires a radical change in diet.

The *loose stool* is always abnormal except when it is the result of starvation or catharsis. It is nearly always accompanied by an increased frequency, or by evidence of indigestion or abnormal fermentation, such as curds, mucus, frothiness, an acid reaction, and often a greenish color. The very loose, or liquid, highly acid, frothy stool that scalds the buttocks is commonly due to an excess, or an abnormal fermentation, of the carbohydrates. The fat also plays a part, though probably secondary to the carbohydrates. A loose, gray, greasy stool means that the fat is not digested normally, or that it is given in excess of tolerance. A stool may be only slightly loose to be abnormal, or it may vary all the way from this to the thin, grayish, spurting, watery conditions found in cholera infantum. *Failure to consider a loose stool as abnormal is one of the commonest causes of lack of success in infant feeding.*

Abnormal Frequency.—Stools in excess of two or, at most, three a day are nearly always abnormal in the artificially fed baby, and are nearly always, at the same time, abnormally loose and acid. The baby with a normal, formed stool, that passes it in three or four installments during the day, is the exception to this rule, but it is also the exceptional baby. *Only after personal examination can a frequent stool be accepted as normal.* The diarrheal stool may vary from two to five, in a mild indigestion; from ten to thirty in a dysentery; up to a practically continuous emptying of the bowels, accompanied by great tenesmus and eversion of the rectal mucosa, in cholera infantum.

Abnormal Size.—Enormous stools occur chiefly with fat indigestion. They are gray or white and excessively foul. They attain their maximum size in Cœliac Disease.

Abnormal Odor.—The normal odor of the breast fed baby's stool is that of fermentation, i.e., acid; that of the cow's milk fed baby, that of putrefaction or decomposition, i.e., cheesy. In the former, the carbohydrates and the fats determine the nature of the reaction; in the latter, the decomposition of the larger amount of casein leads to the characteristic offensive odor. Excessive proteins give varying degrees of cheesy and putrefactive odors, and when the bacterial decomposition of the protein leads to diarrhea, the stools are often excessively foul. If the carbohydrates are in excess of toleration and are abnormally fermented by bacteria, the odor becomes acid, which is always abnormal. The same is true of the fats if they give rise to acute indigestion, or accompany an acute fermentation of the carbohydrates. The large, gray, loose, greasy stool of *fat intolerance* or non-digestion, and the dry, hard, gray soap stool, made up largely of soaps of fatty acids, are both peculiarly foul and offensive. The stool of *cholera infantum* is practically odorless; that of *dysentery* or *ileocolitis* has a relatively inoffensive, stale, musty, mawkish odor, with occasionally that of decay. That of *gangrene* has a dead odor. *Blood*, if present in the stool, gives the characteristic odor of blood; pus, often a faint odor of decay.

Abnormal Reaction.—The normal reaction of the breast milk stool is acid; that of the cow's milk stool, alkaline. Fermentation of carbohydrates and fat leads to acid; decomposition of proteins, to alkaline products, and in cow's milk the proteins are in great excess over those of human milk and dominate the reaction. The carbohydrates, if normally utilized, have little or no apparent effect on the reaction of the normal stool. If in excess, or abnormally fermented by bacteria, they lead to acid stools that are always abnormal in the artificially fed baby. The fats commonly share in this process. Determination of the reaction of the stool with litmus paper is of no practical value, as any degree of acidity that is of significance manifests itself clinically in the looser, sour or yeasty odor, and more frequent and irritating stool. The stool of ileocolitis is commonly alkaline, occasionally acid, depending upon the organism that causes it; that of cholera infantum is commonly acid at first, but usually becomes alkaline later.

Abnormal Color.—The normal color of the stool of the artificially fed baby varies from pale yellow to dark-brown, for reasons already enumerated. To get an accurate idea of the color, the stool must either be fresh, or else crushed and smoothed out with a spatula or between two folds of the diaper, as the outside is often changed from drying and exposure to the air.

The Green Stool.—Except for the greenish-brown of the hunger stool and the cathartic stool, and the occasional pale olive-green of certain normal, formed stools, green is always an abnormal color and is always associated with an otherwise abnormal stool. Green stools are more common in breast fed babies, and in babies fed on high fat and protein mixtures, than in babies fed on simple dilutions, or on fat-free mixtures. Stools that are yellow when passed, sometimes become green *on the outside* on standing. Such a stool is not normal, is practically always hyper-acid, looser and more frequent than normal. The green color is due to a change from bilirubin to biliverdin, caused by some not clearly defined action, possibly bacterial in some cases.

It has no differential diagnostic significance, it is merely an evidence of indigestion or fermentation. It is, however, commonly absent from the most serious diarrheas, such as those of ileocolitis and of cholera infantum. The bacillus pyocyaneus may cause a green stool.

The Pale, Gray or White Stool.—The common cause of this kind of a stool is the overstepping of the infant's tolerance for the fat of cow's milk, in the absence of an acute indigestion, or diarrhea. The stool may be either soft, gray, abnormally frequent and large in amount, and greasy and shiny, when smoothed out; or it may be dry, hard, crumbly, pale gray or whitish, of the color and consistency of putty, as in the soap stool. In either case it has the characteristically foul odor. The white stool may have the same significance as the pale gray stool, merely representing an advanced degree of the same condition. In all of them, however, are varying amounts of neutral fats, fatty acids, and in the soap stool especially earthy salts of the fatty acids. The fat stool reaches its maximum in the so-called Cœliac Disease, in which the fat is not utilizable because of a chronic indigestion probably due to prolonged intolerance to carbohydrates (Howland). When small amounts of iron or bismuth are given, the stool often has a prune whip or grayish color.

In certain diarrheas of relatively rare occurrence, chiefly in babies that are moribund or quite unable to digest milk for the time, and yet are free from marked fermentation, the stools appear as a merely thickened *whitish* or *pale gray yellow milk*, as if the latter had been almost uninfluenced in its passage through the digestive tract, either by the digestive juices or by bacteria.

The gray or white stools above described must not be confounded with the *acholic stool.* The bile in the former is probably present in the colorless form leucohydrobilirubin.[23] The *white acholic stool* is most frequently due to catarrhal jaundice, a rare condition, however, in infancy. If the stools are white at birth and remain so, it means either a congenital atresia of the bile ducts, or an atresia of the intestine distal to the entrance of the common duct.

The Colorless Stool.—In cholera infantum the stool often becomes almost colorless, resembling a turbid serum, or water.

The *black stool* occurs normally only in the form of meconium, or where bismuth, iron or charcoal are given therapeutically. Abnormally it occurs when the feces contain a large amount of blood that has been changed in the stomach from red to black. The white bismuth salt is changed to the black bismuth sulphide by the inter-reaction with hydrogen sulphide. In some diarrheas the bismuth often comes through unchanged, closely resembling white fat curds, probably because hydrogen sulphide is absent in the severer fermentative conditions. It has been recommended that sulphur be given under these conditions to bring about the change to the sulphide of bismuth. The value of this seems doubtful, as the favorable progress of the diarrhea that is usually evident only when the stool becomes gray and then black is probably not so much due to the bismuth sulphide *per se* as to the fact that putrefactive processes are replacing fermentative processes, and this is accompanied both by an amelioration of the diarrhea, and by the normal formation of hydrogen sulphide which naturally reacts with the bismuth salt, changing it to the sulphide. The administration of sulphur assumes that the sulphide and not the bismuth is the active therapeutic agent, and this is difficult to prove.

The *red stool* is most frequently due to an intestinal hemorrhage, and in infancy is most frequently seen in hemorrhagic disease of the newborn. The color varies from light red to dark mahogany, depending on the length of stay in the intestine. (See abnormal constituents of the feces. Blood.) Bacillus prodigiosus may cause a red stool, as may also the administration of carmine, or the eating of beets in the older child.

The *faint pink stain* that sometimes appears after standing, about the margin of the urine in a wet diaper, that contains at the same time a perfectly normal stool, has apparently no clinical significance, and is due to some unknown change in the bile pigment.

The *orange colored stool* occurring in the newborn when cow's milk is given is normal and usual.

Abnormal Constituents. *Curds.*—Two kinds of curds occur in infants' stools—the soft *fat* curd and the hard or *casein* curd. The one is derived from the fat, the other from the casein. They are very different in appearance and consistency, and have a wholly different mode of origin and significance. The duality of curds has been recognized definitely only in recent years[19] (Ante).

The *fat curd* is the only curd that occurs in the breast milk stool, and is the common curd of the cow's milk stool. It is small, soft, white or pale yellow, or greenish, depending upon the nature and color of the stool, and varies in size from a pin head to a small pea. They are made up of neutral fats, fatty acids and their salts, and are quite soluble in ether when acidified and heated. This test is of no practical value, as they can hardly be mistaken for casein curds. Fat curds are formed in the intestine. They are commonly found in diarrheal stools, though they may occur in a nearly normal pasty stool. They are very much more frequent with top milk or cream mixtures than with simple milk dilutions. They represent either a fat indigestion, or perhaps, more commonly, a fat non-digestion, as a result of a carbohydrate indigestion or fermentation.

The *casein curd*, also called the *hard* or *tough curd*, is made up of casein, with a little fat that is purely incidental. Casein curds are formed in the stomach by the action of the rennin and acids upon cow's milk, as we have seen.[7,9] These coagula that are formed normally escape through the pylorus, and under certain conditions are not completely digested and liquefied before they are passed in the feces. As we have seen, curds become increasingly hard. If they pass through the intestinal tract rapidly, due to a diarrhea, the stool curds very closely resemble the milk curds we have seen in the stomach three to five hours after the ingestion of raw milk. If they occur in a nearly normal stool, they have been at least 24 hours in transit and are then smaller and very much harder, not unlike dried, hard American cheese in consistency and color. The former are white or pale yellow, or tinged with a greenish covering; the latter are dark brown or amber colored, and often closely resemble a soaked hulled bean in shape and density. In either case they merely reflect the color of the stool in which they occur. A closer inspection reveals the fact that the coloring is chiefly peripheral, showing that the curds were formed in the stomach before they were exposed to bile in the intestines. In the harder amber colored curds, the color is not wholly peripheral, as all curds tend ultimately to become browner, and it is also probable that the bile stains deeply beneath the surface. A mere comparison of the shape and contour of stool and stomach curds makes it hard to believe that they are not identical in nature and origin. Both are commonly oblong, or oval, or roundish, with a smooth, hard surface indented here and there where digestion has penetrated more deeply. Both present unmistakeably, identical variations in shape, due to the frequent occurrence of bizarre conglomerations that are the result of the tardy agglutination of two or more previously formed curds. When placed in a 10 per cent. solution of formalin, both assume an ivory-like hardness that is characteristic of coagulated casein so treated.

Curds of identical shape, appearance, color and consistency with those just described, are formed, as I have observed,[9] when raw milk is injected into the rectum, the curds varying again with the time that elapses till they are expelled. This merely shows that feces contain a coagulating ferment, as can also be demonstrated *in ritro.* That the "hard curds" are not formed in the intestine, however, is evident from the fact that the milk is known to coagulate before it enters the intestine, and also from Hess'[37] demonstration, that when he excluded gastric coagulation by feeding the milk beyond the pylorus with his duodenal catheter in an infant that was passing hard curds, these ceased to be present after the proper interval.

Hard curds occur to all practical purposes only when raw milk is fed to an infant. The rare exception to this has been discussed in a previous paragraph. Even when raw milk is given they do not occur if the casein is modified in certain ways, and even if unmodified except by therapeutic dilution, they are relatively infrequent. While hard curds are, therefore, undigested casein, they by no means indicate an inability to digest casein *per se.* The same infant that is passing hard curds, that evidently is unable to digest the amount of relatively unmodified raw casein that it is taking, can digest a much larger amount of casein if it is boiled or alkalinized or pre-coagulated, and pass no hard curds nor show any other evidence of casein indigestion. The crux of the matter lies in whether large curds are formed in the stomach. Only from these can hard stool curds originate, and large stomach curds are formed only from raw milk.

Hard curds occur nearly always in an otherwise manifestly abnormal stool. Occasionally the very hard amber colored bean-like curds are found embedded like foreign bodies in nearly normal stools. In my own experience, they never occur in a perfectly normal pasty or formed stool, except in that rare condition where only one or two curds occur that are doubtless the end product of one of those accidentally larger curds that are sometimes formed in the stomach, that even a normal digestion could not liquefy before it escaped into the feces.

While the hard curd probably commonly represents a *casein indigestion,* it is doubtless often a mere *non-digestion* caused by a carbohydrate fermentation which sweeps the normal curds out of the intestinal tract prematurely just as undigested intestinal contents are expelled by a cathartic. Neither is it admissible to consider the hard curd as the only evidence of the difficulty that attends the digestion of the casein, for if there are other symptoms, as seems incontrovertible, these could be present even though the condition were not severe enough to lead to the passing of curds in the stool.

Mucus is present in all stools, for the intestinal mucus glands are constantly active. Under normal conditions it is, however, so intimately intermingled with the rest of the stool that it can be demonstrated only with the microscope or the chemical reagent. It becomes abnormal when it is present in macroscopic amounts. Clinically, the presence of mucus in such amounts means a catarrhal condition of the mucous membrane, and occurs in a variety of conditions. When the mucus appears throughout the stool, it naturally comes from a portion of the intestine above the sigmoid and the rectum, where the contents are still liquid. When it appears as a separate, nearly clear mass, or as a gray coating on a normal stool, it comes from the rectum. Mucus appears abundantly in the *starvation* and *cathartic* stools, and occurs quite regularly in all *diarrheas* from any cause. It is present in large amounts in the stool of *dysentery,* or *ileocolitis,* where it is commonly more or less separate from the fecal matter, and mingled or streaked with blood. In some dysenteries or *ulcerative* colitis it often contains pus. In *intussusception* blood and mucus are commonly intimately and uniformly intermixed without the presence of fecal matter, except at the beginning. At times a teaspoonful or more of nearly clear mucus will be passed with a normal constipated stool, with no apparent clinical significance except a mild catarrhal condition of the rectum.

Blood in the stools is always abnormal, fairly frequent and of great diagnostic significance. The higher its origin in the intestinal tract, the darker the color, due to a conversion of hemoglobin to methemoglobin and hematin as a result of intestinal putrefaction and digestion. If from the stomach it is nearly black or tarry. From this it varies all the way down the intestine through dark mahogany to bright red, if it comes from the rectum. If the blood is copious in amount it means a distinct hemorrhage. This occurs in infancy almost only during the first few days of life in the so-called Hemorrhagic Disease of the Newborn. It may occur in typhoid, but is very rare, especially in infancy. The higher the source of blood, the more intimately is it intermixed with the intestinal contents. If the blood is bright red and separate from the stool, it comes from the rectum. If the stool is hard and is streaked with bright blood, the latter is probably from a crack in the anus. If there is a large amount of bright red blood, especially toward the end of the stool, it is probably due to hemorrhoids, which are, however, rare in infancy.

Blood may occur with any very severe diarrhea, whether digestive or infectious in origin. It comes with or without ulceration. It may occur with *ulceration* from any point from the stomach to the rectum inclusive. Tuberculosis may cause ulceration with bleeding.

It occurs especially in *ileocolitis*, with or without ulceration, and in *intussusception*. In the former it occurs in varying amounts imperfectly mixed with large amounts of turbid mucus, or muco-pus, that is quite separate from the fecal matter that is often more or less normal, except that it is soft and incompletely digested because it is passed through the digestive tract so quickly. In the latter, after the first few movements it is completely mixed with mucus, as a dark, reddish, turbid jelly, and fecal matter is absent. This jelly-like substance follows the withdrawal of the finger after a rectal examination, and this I have seen in no other condition. Occasionally quite red blood is passed, even in intussusception, and especially in the early stage, so that the greater part of such stool is made up of fairly fresh blood.

While blood that is of clinical significance is nearly always evident microscopically, especially if it occurs low down, this is not always so. If there is a suspicion of blood from some higher source, such as duodenal ulcer, where the blood may be slight in amount and concealed by being intimately intermixed with fecal matter, or is perhaps not evident because iron or bismuth, or charcoal, has been given, then its presence or absence is determined by chemical means.

Pus occurs in small amount in ulcerative conditions of the intestine in connection with mucus and often with blood, as in ileocolitis or tuberculosis; in large sudden amounts in a perforating abscess. The same conclusions as to the site of origin of the pus, whether intermingled or separate from the stool, can be made as in the case of blood. Pus that is of clinical significance is practically always evident to the unaided eye. Little can be learned with the microscope, both because pus corpuscles are more or less destroyed by digestion and bacterial action in the colon, and also because polymorphonuclear leukocytes wander normally, to some extent, into the intestinal tract.

Membrane is of very rare occurrence in the stools. It may be found in severe ileocolitis or membranous colitis, and occurs at times in the latter as a cast of the bowel.

Other abnormal constituents are foreign bodies, food remnants, intestinal parasites and their ova, that need not be discusssed here.

Abnormal Presence or Excessive Amount of Food Elements and Their Derivatives in the Stools, as Shown Microchemically.—The Boston School has perfected a microchemical diagnosis of fat and starch indigestion, based, on the one hand, on the relative proportions, or excess, of neutral fats, fatty acids and fatty soaps in the stools, by the use of Sudan III, glacial acetic acid and carbolfuchsin; and on the other, the detection of undigested starch with the use of Lugol's Solution. A detailed description can be found in Morse and Talbot,[23] or Dunn.[27] Such a procedure would seem rather a hyper-refinement of diagnosis that affords no practical clinical information that is not sufficiently evident to the careful clinical observer from an examination of the stool and the baby, without such means. The substitution of a laboratory diagnosis for a clinical diagnosis, here, as in so many other branches of medicine, entails the danger of practical clinical diagnostic atrophy from disuse, especially to the beginner.

Abnormal Micro-organisms.—Except in the case of the different organisms that cause ileocolitis, the bacteriological examination of the stools is in too chaotic and undefined a state to permit of any more practical deductions for diagnosis and treatment than are afforded by macroscopical examinations.

The Feeding of Difficult Cases

This subject will be treated systematically and in detail in a later chapter dealing with the nutritional disorders. There are, however, certain deviations from the normal, that occur very frequently, that can hardly be dignified by classification with the severer disorders, and that might not be recognized in such a classification. They are the usual embarrassments to normal progress that commonly occur in feeding cases that are assumed to be normal until these conditions arise. It is often hard to tell where the normal ends and the abnormal begins, in a subject that is as unnatural itself as is that of feeding babies on cow's milk. From this viewpoint it would seem desirable to consider briefly those conditions that arise most commonly in a sort of practical *proportional sense*, without aiming to present them fully. In ordinary feeding practice, the chief stumbling blocks are vomiting, diarrhea, constipation, the baby that will not gain, the baby that will not eat, and the baby that will not take a bottle.

Vomiting.—*Vomiting must often be considered normal,* or nearly so. Thus the baby that takes an excess of food, either from the breast or the bottle, rather normally rejects a certain amount of it immediately after feeding. Furthermore, the return of food from the stomach is much easier in the baby than in the older individual. Often a slight pressure on the stomach, such as comes from bending the baby forward, from lying on the stomach, from a tight binder, or from laughing, kicking or other over-activity soon after feeding is enough to cause the milk to return. The vomiting that occurs with belching immediately after feeding, too, can hardly be considered abnormal. The stomach normally contains gas, and when this is put under pressure by the ingested food, it normally escapes by the only possible route, the esophagus. If the baby is in the nearly erect position, as when held over the shoulder and patted, the gas alone will escape, often with considerable force, and great relief to the baby. Many babies require such relief after nearly every nursing. If they are not held in the erect position, the gas tends to escape by the same outlet, carrying food before it, often with a gush. If vomiting occurs regularly, except under the conditions enumerated, it must be considered an evidence of either *indigestion* or of *obstruction to the pylorus.* The longer after feeding that vomiting occurs, the greater its significance as a pathological condition.

Of the three food elements that may all cause vomiting from a *digestive standpoint,* it is by no means easy, and often quite impossible, to tell which is at fault in a given case. The *fat* has long been accorded first place in the causation of vomiting, and not without reason. This is due partly to the fact that milk fat becomes peculiarly sour, irritating and offensive in the stomach. It is perhaps due more to the delayed emptying of the stomach, which is caused by and is in proportion to the amount and strength of the fat. For both of these reasons vomiting due to fat is often continued "up to the next feeding" and the returned stomach contents are very sour and acrid. The only practical therapeutic resource is to lessen the amount of fat, by the use of less milk or cream; or by using bottom milk, skimmed milk or buttermilk; or dried or evaporated milk. Homogenization, and the substitution of some other fat, may some day prove useful, but it is much to be doubted whether it will ever be of great practical value. The *sugar* likewise, if excessive, or if in excess of the infant's tolerance, will also cause vomiting. The *casein* is unquestionably a factor in producing vomiting if given in excess, and in raw milk, for we know that the best weapons against the vomiting of raw milk are dilution, boiling, alkalinization, peptonization, the use of cereal waters, pre-coagulation with lactic acid or rennin, etc., all of which have a marked effect on the curd, and so far as definite knowledge goes, no other effect. The favorable result that comes in these cases, when evaporated or dried, or condensed milk is substituted for fresh raw milk speaks for the same thing, though it is possible that the process of drying or condensing has some effect other than on the casein, as, for example, on the fat.

The common statement of mothers and nurses that the vomiting is not bad because "the milk came up just as it went down, not sour or curdled," has no clinical significance, except that the baby vomited immediately after nursing. If it vomits later "sourness and curdling" are not in themselves abnormal, because all milk curdles in the stomach after a short time, and sours characteristically after two or three hours.

If in spite of employing all these measures to combat vomiting, i.e., reducing the fat and sugar; modifying the casein; lessening or lengthening the feeding interval; with a proportional lessening or increasing of the amount of food; and especially the use of mother's milk; the baby still vomits, then one is justified in reaching the conclusion that we have a *pyloric obstruction.* If the obstruction is not so complete as to interfere with reasonable progress, one must state promptly that such a condition exists, and that for a time the baby will continue to vomit and not do so well, but that it will ultimately overcome the condition. If a true *hyperplastic stenosis* of the pylorus exists, with such a degree of vomiting that dangerous inanition is inevitable, and especially if a pyloric tumor is definitely palpable, then feeding alone will probably no longer lead to results, and the surgeon must be called in.

Diarrhea.—Vomiting is commonly an earlier and milder symptom of a digestive disturbance, diarrhea, a later and more serious one; usually the two go together, but by no means always. By diarrhea, here, is meant simply an increased frequency and looseness, and commonly acidity of the stools. There may be only two or three loose, sour stools, or there may be a great many. The diarrhea may be due purely to an indigestion, or to an indigestion plus bacterial fermentation, or it may be primarily a bacterial fermentation with an accompanying non-digestion. It is by no means easy, nor even possible many times, to tell which condition is primary or predominant. If there are only a few undigested stools, without excessive gas or sourness, and there is no temperature, we have to deal probably with a simple indigestion. If the stools are more frequent, are acid enough to scald the buttocks, are frothy and spurting, and there are rises in temperature, then we may feel sure that we have no longer a simple indigestion, but a bacterial *fermentation* that now dominates the picture. If the stools are frequent and foul, but not acid, the whole thing may be due to rarer predominatingly proteolytic bacterial action rather than to the common fermentative action on the carbohydrates and the fats. Fortunately, a finer distinction between an indigestion, on the one hand, and a bacterial condition, on the other one, is of no great practical value, as the treatment is practically identical.

The role of the different food elements in the causation of these diarrheas, whether they are primarily at fault, or only secondarily, as a result of bacterial activity, must always be considered in each case, though it is again often impossible to determine which food element or combination of food elements is responsible. In cases of any severity, a therapy based on reduction or modification of *one* food element rarely leads to success. A reduction or discontinuance of all food for a brief time is necessary, to be followed by a gradually increasing amount of food always short of an amount that will bring on an aggravation of symptoms.

The *carbohydrates* seem to play the leading part in the diarrheas of infancy, as they are the only food element that readily undergoes fermentation. This applies only to the artificially fed infant, for in the breast fed baby, fermentation is normally the predominating process, and in the medium of breast milk, sugar is naturally tolerated in large amounts. In the bottle fed baby the carbohydrates must be carefully watched and carefully dosed, and especially so the moment there is the slightest tendency to diarrhea. In the normally constipated baby, sugar is tolerated in large amounts, no matter what the kind of sugar may be; and in general, the less the amount of fresh milk, the greater the tolerance for sugar, and *vice versa*, a fact that must always be borne in mind in practical feeding. Whenever there is a tendency to diarrhea, the dextrin-maltose preparations, and especially those relatively high in dextrins and low in maltose, are tolerated in larger amounts than are cane sugar and milksugar. Starch in normal conditions in therapeutic amounts is constipating as compared with sugars. In diarrheas it requires the same vigilance, in larger amounts, as do the sugars, though in older children after the sixth month it is commonly much better tolerated than the sugars. In smaller amounts, starch is apparently always tolerated, as is evident from its wide use as barley water as the sole food in diarrheas, where therapeutic starvation is indicated. That it has here any constipating effect, as is commonly believed, is certainly open to serious doubt, as this view disregards the fact that the important therapeutic measure is the temporary withdrawal of milk, not the use of barley water. The use of barley water as a temporary sole food is perfectly justified, because when given alone it does no harm, and it supplies a small amount of food that may avoid a serious catastrophy that might follow complete carbohydrate starvation, especially if improperly prolonged. That it should be given without sugar when so used would seem to be self-evident. A grain of saccharine to a quart of barley water will commonly make the latter wholly acceptable to the baby, where the unsweetened food would be refused.

The *fats* of cow's milk unquestionably play a large part in the upkeep, if not in the causation of diarrheas. While normally constipating when given nearly alone, or in the absence of sugar and much whey, as in the use of Eiweissmilch, they are readily involved secondarily in any diarrheal condition, especially when fresh raw milk is used. The fat is in every way closely allied to the carbohydrates, but is probably only involved in the fermentative process that leads to diarrhea if there is a primary carbohydrate fermentation. Even the fat curds that are given all together too much weight as an evidence of a primary or predominating fat intolerance may be simply an accompaniment of a carbohydrate fermentation, or of any diarrhea of any origin which acts merely as a cathartic would to sweep out the intestinal contents prematurely. Fat, at best, is never completely utilized. In general, the fat of cow's milk cannot be given as freely in early infancy as can that of breast milk, and must be used cautiously throughout infancy, not only to avoid *fat constipation* in certain combinations, but also to avoid fat diarrheas in other combinations that are readily fermented. In diarrheas the fat must be materially reduced, or even eliminated, as in the use of buttermilk or fat-free milk, except in the use of Eiweissmilch, where the sugar is low and the protein, which is antagonistic to fermentation, is high. In any combination, and especially in fresh milk, reduction of the fat must be accompanied by reduction of the sugar for the time being.

The *casein* unless carefully safe-guarded, as it is *ab initio*, in nearly all of our milk modifications, is a prolific cause of diarrheas, with or without the presence of hard stool curds. While protein which normally undergoes putrefactive changes in the intestinal canal has innately a constipating or anti-laxative effect, both because it is completely absorbed, and because proteolysis is an inhibitor of saccharolysis, the casein is nevertheless a cause of diarrhea when given in sufficient amount in raw cow's milk. This is evident alone from the fact that raw milk diarrheas are stopped by boiling the milk or adding sodium citrate, which affect in this respect, so far as our definite knowledge goes, only the casein by making it finer and more digestible. Even more convincing is the change to evaporated or dried or condensed milk, where the curd is at the minimum and the therapeutic effect at its maximum. That the casein curds in raw milk indigestion act as a mechanical irritant, as is suggested by the occasional violent symptoms that arise from eating an excess of corn, grapes, peanuts, cucumbers, unripe apples and other foods that leave large undigested and undigestible masses in the intestinal tract, is possible and even probable, but has not been demonstrated, and is not necessary to the assumption that in some way the casein acts as a disturber of digestion, with a resulting diarrhea. The most convincing effect comes from the fact that Eiweissmilch in which casein curds are not exceedingly fine, acts as an irritant and not as a healer in diarrheas.

That the casein of cow's milk rarely causes diarrhea or any other marked digestive disorder, is true, but it is true only because its harmful effect is forestalled consciously or unconsciously by nearly every therapeutic device that we use as a routine in modifying cow's milk for babies. When we use these the casein need give us no concern, even in extra-therapeutic amounts.

The *salts* may play a larger part in the causation of diarrhea than is now known. The Finkelstein school has attracted considerable attention to them in this connection, and in Eiweissmilch the whey salts are greatly reduced. Meyer's Molkenaustausch[6] would point to the same conclusion, as would also the use of *whey reduced* and *whey adapted* milk. A modification of the salts in this connection is not of practical value except in the use of Eiweissmilch, and here other factors becloud the issue.

In the *treatment* of the *simple diarrheas* that occur in connection with the ordinary feeding cases, it is often impossible except in the milder forms to tell just which food element is at fault, or to gain a decisive therapeutic result by a modification or reduction of that food element alone, unless it was greatly in excess. When a diarrhea is well established or moderately severe, it becomes necessary to reduce all of the food very greatly, and in all severe cases to eliminate it completely for 12 to 24 hours. During this period barley water, or tea, or water with saccharine, if necessary (1 gr. to the quart) can be given. If the condition is not too severe, and especially if the baby is in the second semester of life, the same food can gradually be restored, the sugar more slowly than the milk, which is best boiled and alkalinized, and always under careful control as to the number and nature of the stools. The dextrin-maltose preparations are here most serviceable, except that in the older infant starchy preparations like flour ball, Imperial Granum, farina and zwieback are often more quickly tolerated in larger amounts than is any form of sugar. If the diarrhea is at all severe it is best to abandon ordinary fresh milk modifications all together for a time, and resort to some more easily digested food. The younger the infant the more imperative it is to make a radical change. Mother's milk is always specific and if results are not obtained quickly and decisively in the first month or two of life, it is best to lose no time in getting breast milk. Only a small amount in connection with other food will often turn the scale. Next to breast milk must be placed Eiweissmilch. When this is not feasible because of the difficulty of making a good preparation, or of procuring either a fresh preparation at a diet kitchen or a dried preparation (such as Hoos' Albumen Milk), then the use of dried, or evaporated or condensed milk or buttermilk especially commend themselves as temporary foods to be used in cautiously graduated amounts closely controlled, until a normal condition has been established and maintained for some time. The change to fresh milk should then be made suddenly, not gradually, always beginning with a smaller amount than the utlimately desirable amount.

Constipation in the artificially fed baby has been given an undue prominence, chiefly because it has been viewed from the adult standpoint, and also because mothers do not appreciate that the normal loose breast fed stool, to which they have been accustomed, is not the standard of what is normal in the cow's milk fed baby. As we have seen before, cow's milk is constipating both to the adult and to the infant if it does not cause, especially in the latter, an acute disturbance with diarrhea. A formed hard stool, or a dense smooth pasty stool, is normal in the artificially fed baby, and becomes abnormal and requires treatment only when there is failure, or difficulty in passing it, as when it is excessively dry, pale and foul, and is accompanied by a failure to gain in weight. Other conditions that may cause constipation are painful anal fissures and hemorrhoids; pyloric stenosis; an overtight anal sphincter; relatively large colon and sigmoid flexure with abnormally long mesentery; weakened abdominal and intestinal musculature due to rickets and other forms of mal-nourishment; failure to teach the baby to make the effort to have a bowel movement at a regular given time; the excessive use of cathartics, expecially castor oil, of drastic suppositories, such as glycerine suppositories, and of soap and glycerine enemata; and finally, the rare occurrence of tumors, or chronic peritonitis, or adhesions.

In general, the treatment depends upon both the *cause of the constipation* and the *nature of the stool.* While emphasis is rightly placed on *dietetic treatment* that has become almost a fetish with many laymen, it must be said that some cases in the first half year of life, especially, will not yield to any form of dietetic treatment that is not capable of more harm than are the simple non-dietetic measures that must often be resorted to. In general, dietetic measures consist in reduction of the milk and increase of the carbohydrates. The former should never be carried to the point where the infant's protein balance is endangered, somewhere between 1 and 1½ oz. of milk to the pound of baby, and the latter should not be carried beyond the child's sugar tolerance. The carbohydrate is best given in the form of dextrin-maltose, which is tolerated in larger amounts than other sugar, and if constipation still persists, a preparation in which the maltose is relatively high is preferable. The standard malt extract preparations are here often serviceable, but must be used cautiously because of their decided laxative action. In cases of fat constipation with soap stools (Milch-nährschaden), these two measures must be carried to the maximum, and for this purpose Keller's Malt Soup was devised and is most serviceable. In the older baby other carbohydrates, such as the coarser cereals, and also vegetables and fruits that give to the baby what veterinarians call "roughage," help to replace the milk in part, also to promote peristalsis by making a larger stool, in which, too, fermentation plays a relatively greater part, as in those of the herbivorous animals. Prune juice early, and later prune pulp and whole prunes and stewed figs have a popular and apparently well-founded reputation. Olive oil is of relatively little help. Orange juice can be given very early but has very little laxative action, if any. The addition of cream or top-milk may help or may aggravate the condition, depending upon the baby and upon its state of digestion.

If, in spite of dietetic measures, the baby still has no natural stool, or one that is too hard to pass without too great difficulty, then one must resort either to *cathartics* or to *suppositories* or *enemata*. This need cause no concern, for if properly and rationally carried out they are not harmful now, nor do they promote future constipation. If the stool is hard and difficult to pass, a mild *laxative* should be given regularly. If it is soft, or not hard enought to pass with difficulty, it is best to stimulate the rectal reflex by the introduction of a mild *suppository*. Probably the best laxative is milk of magnesia, which can be given in one bottle, usually preferably in the night bottle. From one-half to two teaspoonfuls can be given. If this leads to griping when administered in the necessary dosage, as it sometimes does, then a liquid petrolatum is often the most serviceable remedy. From one to three or more teaspoonfuls a day given between meals will keep the stool soft, and makes it easy to pass, unfortunately, at times, too easy! The most frequently used laxative for children and babies is Castoria, against which there is apparently no objection except that it is expensive, patented and of unknown composition. Castor oil has no part in the treatment of constipation, but the leading part in that of diarrhea. Because of its constipating after effects, it only aggravates the former, but acts most favorably in the latter.

If a laxative is used it should be given, not occasionally to meet an urgent condition, but daily so that the habit can be formed of having a movement regularly, and so the stool will never become hard and painful to pass, which in itself is a potent cause of constipation in an infant. The amount can gradually be lessened as the habit is formed, especially when aided by careful training of having a movement daily at a fixed time.

If the stool is *soft*, or at least not hard or painful to pass, laxatives would hardly seem indicated. One has here, possibly, some mechanical factor referable to the colon or sigmoid flexure, or an overtight sphincter or an atonic condition, though commonly no cause can be established. This is seen at its best in the breast fed baby that one encounters not infrequently whose stool is soft and mushy, but is passed only with assistance. Some form of rectal stimulation is here clearly indicated, which should be as mild as possible. Sometimes merely tapping over the anus with the finger is sufficient. Commonly all that is necessary is the introduction of a foreign body that stretches the sphincter muscle, and the mother who dips her little finger in oil and uses it for that purpose has, at least, a therapeutic measure that meets all requirements ideally. Glycerine suppositories are clearly contra-indicated, as are soap-sud enemata, in cases of chronic constipation, for they accustom the baby to a stimulus that will soon cause it to pay no attention to the normal stimulation caused by feces in the rectum. The same is true in a lesser degree of the soap stick dipped in warm water. If covered with oil or vaseline, the effect of the soap is eliminated and it commends itself most highly.

Occasionally one sees a baby in which a condition of severe constipation is evidently due to a *tight sphincter*. In one such case in a newborn baby, that clinically resembled a Hirschsprung's Disease, with an enormous tense distention of the colon, and almost complete inability to pass even gas, except about once daily, with a loud explosion, the condition was wholly reduced permanently in a few days by the gradual introduction of a larger finger each day for a number of days, with an occasional repetition of the treatment by the mother for some months.

In *fissure ani, hemorrhoids* and other conditions, the appropriate treatment discussed elsewhere should be carried out. It is most important that the movements be kept soft and easy to pass, and here again liquid petrolatum is most serviceable. If other laxatives are given care must be exercised not to give them to excess, for tenseness and straining naturally aggravate the condition. In rickets, malnutrition, weakness and all acute febrile and other conditions accompanied by a lack of appetite and resulting constipation, the indications are to remedy the underlying condition, and for the time being to treat the constipation along the lines indicated above. Abdominal massage along the course of the colon is said to be of value in so-called atonic conditions.

The Baby That Will Not Gain

A normal gain in weight is the one most tangible evidence of normal progress. Failure to gain means in reality a loss and ultimate malnutrition. It is commonly necessary to keep a young baby's food low to avoid an acute upset. The mother must often be told frankly that her baby will not gain much for a time, that the most rapid progress can be made by proceeding cautiously and avoiding digestive disturbances, which always make the baby harder to feed for some time after their occurrence. Some babies will, however, fail to gain properly even after the first few months and after receiving an adequate amount of food. These babies usually fall readily into two classes, those that are obstinately constipated, and those that have a tendency to diarrhea or, at least, looseness of the bowels. The constipated baby usually yields readily to rational treatment, for constipation admits of wide latitude in the amount and variety of food. The baby with a tendency to diarrhea must commonly be underfed for a time on a very restricted choice of food, unless one at once proceeds to breast milk, or to one of the special foods devised to meet this indication, such as Eiweissmilch.

The **constipated baby,** especially if it has a hard, dry, gray, or pale yellow stool, has usually exceeded its tolerance for milk. Increasing the milk in the hope of bringing about a gain only makes failure more certain. Unfortunately, just these cases, especially after the fifth or sixth month, because they have not done well, are kept all the more on a milk diet because of the fear of so-called "solid food." The rational treatment consists in lessening, or at least not increasing, the amount of milk, provided it is sufficient in amount to cover the child's needs, and increasing the carbohydrates. It is of distinct advantage to add several carbohydrates, starch, dextrins, sugar in various stages of conversion, because a larger amount can thus be tolerated. In the young baby, if the condition is not serious, the addition of a cereal water is often enough to produce a change for the better. As we have seen, the addition of starch brings about a favorable condition that cannot be explained wholly by the simple increase in food. In all but the mildest cases it is necessary to increase the carbohydrates often beyond the usual amounts. It is here that the dextrin-maltose preparations are most useful, both because we are adding two carbohydrates and because they are more fattening and are tolerated in larger amounts than are cane or milk sugar. In the severer cases Keller's Malt Soup with its maximum four-fold carbohydrate, and minimum milk content, best meets all of these indications. In the infant over four months old the same result is obtained by feeding it like one would an older child, by giving it not too much milk and adding farina, cream of wheat, oatmeal, zwieback, cracker or toast, broth, fruit juices, and after the fifth month, finely divided vegetables in vegetable soup. The therapeutic result thus obtained is one of the most rapid and decisive in all infant feeding. In the younger infant that will not gain on any ordinary modification of fresh cow's milk, one must either worry along until older food can be given, or else proceed at once to a food rich in carbohydrates and low in milk, like malt soup or condensed milk, or to a food like buttermilk or acidified milk, or dried milk, with which a larger amount of carbohydrates can be given than with fresh milk.

The baby that will not gain because of a **tendency to diarrhea**, presents a much more difficult problem. With fresh milk mixtures, unless kept below the point of maintenance, the baby has one acute disturbance after another whenever the food is increased in any way, up to a point necessary to insure a gain in weight. This means a loss, due not only to the digestive disorder, but also to further therapeutic starvation and the increased difficulty in returning to a normal condition. The treatment has been given in a preceding paragraph on diarrhea. The younger the infant the greater the indication for breast milk. In any case, it is best to abandon fresh milk after a fair trial and proceed at once to dried, evaporated or condensed milk, or to buttermilk, or, better still, to Eiweissmilch, until the stools have become normal and have remained so for several weeks.

In the second or third year of life one occasionally finds a baby that has a distinct chronic tendency to loose, sour, over frequent stools, with an accompanying failure to gain in weight. These cases present an embarrassing problem because it is so hard to find enough of an appropriate food to give them. The rational treatment consists in cutting down to the necessary point the foods that ferment, such as the sugars, fats, and especially the starches, and substitute for them to a large extent the foods richer in protein. Fresh milk is commonly not well borne, no matter how modified. Dried or evaporated milk or fat-free buttermilk, together with scraped beef, broths, finely divided or sieved vegetables with a small amount of zwieback or toast should constitute the chief diet, until there is a return to a normal stool. Fruits should scrupulously be avoided for some time, except orange juice, which can commonly be resumed early.

The **child that "will not eat"** represents probably 10 to 20 per cent. of children in private practice between the second and fifth year inclusive, with a lesser incidence in the first year. In the *young infant* it commonly means an excessive milk diet, with fat constipation, and a resultant disturbance of well-being. The treatment for this has been indicated above. Occasionally, it means that the baby has become tired of a food that is too sweet, or has too highly specialized a taste, such as that given by dextrin-maltose preparations, when used in the enormous amounts called for by the directions that accompany the food. Here often a change to a lesser amount or to a different sugar will bring about a return of appetite. Often though the cause is not apparent. In these often a brisk cathartic, a day of barley water, and a short period of restricted food will restore a lost appetite. In some babies, however, neither is the cause apparent nor the remedy at hand, and it becomes necessary to worry along till the time comes when they will perhaps take more kindly to spoon food. The addition of a little more salt to the food would seem indicated at a time when only liquids are given, to increase thirst, with the hope that it will be satisfied by the liquid food. Babies here, as in febrile conditions, seem however to make a sharp distinction between water and any liquid food when a relief from thirst is looked for.

In the *older child* the trouble lies not in the food but in the child, his environment, and his mode of living. It is *psychologic* rather than physical or physiologic, a natural result of the abnormal, constantly restrained, nerve racking, high tension, cooped up existence to which the city child is subjected, which finds its highest development in the modern flat in a crowded district. A certain proportion of these children were not given spoon food and coarser foods and a proper variety of food at the proper psychological time, the fifth or sixth month. Such children are "picky," will eat only a few dishes and have a fondness for soups and a dislike for cereal, and especially for vegetables. The condition is aggravated by over-urging, by scolding, coaxing, cajoling, and bribing and by enforced feeding often till the child vomits either from necessity or because he has learned that this is his only effective weapon against further stuffing. Ludicrous measures are often resorted to. In one of my cases the mother would slip the food into the child's mouth, the grandmother sitting by and all prepared would suddenly beat a drum, the child would become so interested in the noise, that it allowed the food to slip down instead of spitting it out again. Combined with overurging there is commonly an endless family discussion in the presence of the child, as the battle is renewed at each meal, until the child finally comes to look upon himself as an unusual child, sort of *sui generis*, "who will not eat." A further factor, I am convinced, lies in the innately cruel and tantalizing attitude of young children who take delight in paining others, as, for example, here by not eating. All of this reaches its maximum in the one child family, that sad exotic outgrowth of civilization. In this parental solicitude reaches a pathologic development. In this latter case aloneness during meals, or, as among the wealthy, with only an older nurse who is the constant monotonous companion day and night, makes for loss of appetite, just as the crowd makes for appetite at the banquet, or the barracks. The single child commonly does not eat well, the orphan asylum child eats everything set before him, just as his companions do. In the large old-fashioned family, not eating was rarely a cause for concern.

The remedy for not eating, from the very nature of the cause, is manifestly difficult to obtain. Tonics, like cod-liver oil and iron, do very little if any good, for the condition is not physical. Neither does fresh air nor exercise have much influence. Often these children look well nourished, evidently getting enough food one way or another, commonly by eating something between meals, which only enhances the condition but relieves the distressed parents for the time, at least. *The only effective remedy is a complete change of environment, with elimination of the mother or nurse to whom the child has been accustomed.* If the child must remain in the household, eating at the table with the family, or in the kitchen with the maids in wealthy families, instead of alone with only his mother or nurse sitting beside him urging him to eat, is sometimes effective. I know of one mother who took her child to a new restaurant each noon, with good results. The effect of change of environment is seen in the child who will not touch carrots at home, yet will ask for a second helping at his grandmother's, or who "will eat nothing at home" but will cause consternation to his anxious mother by his voracity at a picnic or a children's party. I know of a child of five years, with an English nurse as a constant companion, who begins to wretch when her breakfast is brought in, and almost invariably vomits before she is through. When the family took her away for the summer to a new environment, she ate normally and gained rapidly, only to sink back to her former condition after being home for a week. A normal association with other children of the same age, constant not intermittent, instead of with older, monotonously uninteresting people, often brings about a more normal condition. In a one child family the employment of a maid with a child of the same age led to an immediate result. The effect of both change of environment and of more normal association with other children is perhaps best illustrated when such a child is taken to a hospital ward. I recall one such child of about eight years of age, of Jewish parents, in whose home all of the factors that make for not eating, including the absence of brothers and sisters, existed in the highest degree. The mother stated that "he would eat nothing and would always vomit if he did eat anything." The condition seemed so severe, from her description, that a good clinician suspected a pyloric or other obstruction and sent him to a hospital ward. During the two weeks that he was in the hospital, he eat everything set before him, did not vomit once and gained rapidly. The problem of the child that will not eat must be met in each individual case along the lines indicated above, but unfortunately the solution is often not available.

The baby that will not take a bottle deserves more than the casual mention that is given the subject in all of our text books. Refusal to take the bottle when mixed feeding is begun is extremely common, is peculiarly annoying, not without embarrassment to normal progress, and at times even dangerous. It is withal easily and absolutely preventable. That information on this phase of the subject is sadly wanting is evident from the fact that this symptom is so common and that practically nothing is being done to prevent it. With constant and painful regularity one hears the same statement, "my baby will not take a bottle," often embellished by, "none of my babies would ever take a bottle," as if it were characteristic of the special breed. Physicians, too, seem to accept the explanation of an *individual peculiarity* instead of recognizing it as a perfectly normal phenomenon that at a certain age a baby will refuse a bottle if it has not been accustomed to it. The whole subject might seem trivial if it were not for the fact that refusal to take the bottle takes place at a time when drinking from a cup is only exceptionally successful, and feeding from a spoon unsatisfactory, and commonly quite inadequate. Clinically, the subject usually presents itself in the following manner: A mother who has had an abundance of milk up to the sixth or seventh month decides to give the baby one bottle a day. To her dismay she finds that he will not take it. She tries various mixtures, including a number of "infant foods," and various nipples, even the wide-mouthed bottle with its dome-shaped, breast simulating, nipple, all with no result. She now recalls that former efforts to give him water from a bottle were unsuccessful, and decides that he is not yet ready for a bottle and puts off the time for trying it again. A new trial leads to the same result, and the matter is put off again, till the proper psychological time that will never come. In the meantime, the baby is underfed at the breast, and losing. Panic stricken, her milk leaves rapidly. Finally, through an error in interpretation, she weans him abruptly "so that he will take the bottle," only to find him as obdurate as ever. Frantic efforts to make him take the bottle, "spending hours at a time," "holding his nose," etc., only sharpens his obstinacy, and a day or two of starvation leaves him unchanged, only weaker and more determined. The spoon and the cup are now brought in with. only too frequently, slow and painful progress. I have seen at least one death from this cause, and several narrow escapes. The common result is the pathetic change from a fat, rosy, blooming baby, of the first half year, to a thin, pale, under-nourished, unhappy baby of the latter part of the first year. The condition is all the more pathetic when we contrast it with the thin, scrawny, crying, artificially fed baby of the first semester, that becomes rosy and fat and strong in the second semester, and we realize that a wholly preventable error in technique caused this condition in the breast fed baby.

The *prevention* of this unhappy sequence of events is very simple. *Every baby should be accustomed to a bottle from the first weeks of life, without ever a day's omission.* The baby of eight or nine months that refuses a bottle, if he has not been accustomed to it, does so, not from lack of hunger nor disinclination to eat, nor a dislike for the food offered, but because he has learned only one source of food supply, and the bottle does not suggest food to him. The reverse is equally true, the baby that has taken only the bottle will as resolutely refuse to take the breast at the same time. That it is not absence of hunger that makes the former refuse the bottle is evident from the avidity with which he seizes the breast; that it is not the nature of the food, from the fact that he will refuse his mother's milk if put in a bottle. Commonly all of this happens only to mothers who have an abundance of milk for at least six or seven months. The baby in this way gets an adequate amount of water, is even surfeited and so refuses the water offered him in a bottle, even in early infancy. This is not surprising when we recall further that babies are peculiarly fond of sweet things and that mother's milk contains 7 per cent. of sugar. The baby can at this age easily be induced to take a bottle of water, *for the purpose of accustoming him to it, if a little cane sugar is added to it,* and against this little addition of sugar to the enormous amount he is getting from his mother, there can be no valid objection. Only a small amount need be given each day to keep up the habit thus formed.

The refusal to take the bottle has a *fairly constant* and very *interesting chronological setting* that must be taken into consideration in the *treatment*. In the first three months of life there is practically no trouble whatever in passing from the breast to the bottle, or from the bottle to the breast. In the fourth, and especially the fifth month, one begins to encounter some difficulty, that can however nearly always be overcome without discontinuing the accustomed mode of feeding. In the sixth or seventh month the difficulty greatly increases, and by the eighth month the chances are no longer even that the baby will make the change. The ninth month usually sets the time beyond which it is no longer possible to induce the exclusively breast fed baby to take the bottle. There are exceptions to this, but they constitute the great minority. After the sixth month it is often of value to stop the breast feeding and to absent the mother; before that time it is rarely necessary. This expedient usually fails after the eighth month. At any age no attempt should be made to force the bottle, or to starve the baby into taking it. The former is merely resented and the latter is based on a wrong hypothesis. Trying all kinds of nipples is of equally little value. Rather, the bottle with the food sweetened to taste like breast milk, commonly preferable with cane sugar, with a large hole in the nipple so that the food escapes into his mouth without effort, should be offered often, rather in play than under compulsion, before nursing, during nursing and after nursing and between feedings, when hungry and when full, with the hope that in some favorable time and mood he will once draw and learn this new method of taking food. If he once takes an ounce, the battle is won.

If the baby cannot ultimately be induced to take a bottle, then he must be fed from the cup and the spoon. If the condition of undernourishment is severe, and even these are refused, then one must resort to the medicine dropper and even the stomach tube. Often a good deal of milk, which is best given as concentrated as expedient, can be soaked up in cereals or zwieback, or cracker, or given in the coagulated form of junket, and in a custard.

SPECIAL FOODS TO MEET SPECIAL INDICATIONS IN FEEDING DIFFICULT CASES

The normal baby that has no digestive disturbance can commonly digest fresh cow's milk and make normal progress. Some of them, however, cannot, especially in the first few months of life, and if they have once had a digestive disturbance with diarrhea, it is often hard to get them back on an adequate amount of fresh milk. In these cases it is commonly advisable, while not absolutely necessary, to make a radical temporary change to a food more easily digested, under the circumstances, and to continue this until normal digestion is again established and the baby can again be returned to fresh milk. How early to turn to one of these foods, or in what class of cases to use them, whether before or after trying to adjust fresh cow's milk, must remain with the experience of the clinician. Some of these foods are very similar in action and fundamental composition, though superficially they may seem very different. Which one the individual clinician will choose depends largely upon his peculiar experience, which often goes back to some early successful use of a particular food or method. Practically, it is of great value to have some favorite procedure, some favorite food; to perfect one's self in its use, rather than to choose constantly the new idea, that is commonly an old one disguised in new clothes, or with a new name, for they all lead to the same result.

In general, I am convinced that in the future we will more and more reach the conclusion that fresh cow's milk is hard to digest by the baby, and that if after a short, reasonable trial no progress is made, we will quickly abandon it for the time being and turn to one or the other of the foods about to be discussed. To worry along under the guise of science in an attempt to foist a food upon a non-receptive baby, when one can give it something that will commonly almost immediately restore it to a normal condition, and that will insure normal progress for a time at least, is not only unwise, but does not even give one the consolation of being "scientific," because it lacks the essential attribute of common sense.

1. Dried, Evaporated or Condensed Milk.—Condensed milk was first introduced by Gail Borden in 1856. Out of this process of condensing and canning milk, so that it can be transported more easily and kept indefinitely, has grown an enormous industry now mounting to many hundreds of millions of dollars annually in this country alone. Various modifications of this original product have been put out since, such as evaporated milk, dried milk, and a host of other brands of condensed milk. Especial interest has centered on all of these in the last few years, because of the necessity of sending milk to European countries during and since the war. Various methods of condensing milk are employed that need not concern us here, but that may have some as yet undetermined bearing on these products as infant foods.

By "*condensed milk*" is commonly meant milk evaporated to one-fourth to one-third of its bulk, depending upon whether whole milk or partly skimmed milk is used, to which is added nearly an equal amount (about 40 to 45 per cent.) of cane sugar. By "evaporated milk" is meant a milk condensed to the same degree, but unsweetened and sterilized. By "dried milk" or milk powder, is meant a milk, or skimmed milk, reduced by rapid high heat and evaporation to a dried form, without additional sugar. The chief representatives of this are, Dryco Milk (formerly Honor Brand) and Klim, the former of which is most used in infant feeding. Mammala is a dried milk from which about one-half the cream has been taken, and milk sugar added up to about 55 per cent. The following table compiled from various sources gives the approximate composition of the three types of milk:

	Condensed milk, Eagle Brand	Dried milk, Dryco Milk	Evaporated milk
Fat	9.61	12.00	11.00
Protein	8.01	34.00	10.00
Milksugar	12.03	44.00	13.00
Cane sugar	42.91	0.00	0.00
Ash	1.78	7.00	2.00
Water	25.66	3.00	64.00

The use of condensed milk as an infant food has been discussed under "Proprietary Infant Foods." *Dried milk* belongs in a quite different class. It is simply milk or partly skimmed milk reduced to a powder form, which requires only the addition of a given amount of hot water to make a mixture of the same composition as fresh milk. This mixture can then be modified by the addition of carbohydrates, as is done with fresh milk. In practice, it has been found that the amount needed by thriving babies is somewhere between two and two and one-half level tablespoonsful per pound of baby, daily, depending upon the amount of sugar and other carbohydrates used. In digestive disturbances, one begins with a small amount and proceeds to the larger amount, as the baby will tolerate it.

The advantages of dried milk are so obvious that it has become increasingly popular in the last few years. That dried milk is more easily digested by the baby that does not digest fresh milk satisfactorily, or by any baby, can be doubted or denied only by those whose prejudices have not permitted them to use it. Just as boiled milk is easier to digest than raw milk, so dried milk is easier to digest than boiled milk, and probably for the same reason. They can unquestionably be given quicker and in larger amounts than can fresh milk, and this is of great importance in the baby that is under-nourished and continues to be so, if kept on a too restricted or too undigested a fresh cow's milk mixture. Other advantages of dried milk are its easy transportability and the fact that it keeps a long time even after the can is opened. It can, therefore, be used as needed in the household, in occasional supplementary feedings, on long journeys, in regions where fresh milk or pure milk cannot be obtained, and in many other ways and places which suggest themselves.

The greatest advantage to the physician lies in the fact that he can avail himself in feeding babies of the well established, easier digestibility of condensed or dried milk, as compared with fresh milk, without becoming *particeps criminis* in the use of a most objectional Patented Baby Food. Much as I would prefer not to say it, I am convinced that dried milk is, however, not as easily digested as condensed or evaporated milk, though more so than boiled milk. The difference between the two types of milk is, however, not so great clinically that it is not commonly over-balanced by the dangers that exist in using a food so peculiarly objectionable from every other standpoint as is condensed milk. Condensed milk is a safe food for a very short time only, and just because it is so easily digested it is peculiarly dangerous, because the mother is apt to continue it for that very reason. Dried milk, on the other hand, is a safer food to continue over a longer period of time. It is simply a convenient *sterile* dried milk that is easy to digest, and has apparently lost little of its antiscorbutic properties, and there is, as yet, no evidence that it has a much greater tendency to produce rickets than has fresh milk.

While fresh cow's milk can be accepted as the present best substitute for mother's milk, it leaves so much to be desired that we are all looking for something better. I am convinced that in the future some form of dried or evaporated milk will have an increasing usefulness in the feeding of babies, a usefulness that may far exceed our present expectations.

2. Precoagulated Milk. Buttermilk; Acidified Milk; Pegnin or Chymogen Milk; Eiweissmilch.—In one form or another, milk that has been coagulated and then beaten up into a liquid form has been a favorite food for delicate babies, and especially those with a tendency to diarrhea. In one form it is coagulated by acid formed by lactic acid producing bacilli, and is called buttermilk, or acidified milk. In another, it is coagulated by pegnin or chymogen, or some other form of rennin preparation. In the preparation of Eiweissmilch, both of these methods are used, together with the elimination of a large part of the whey. To what extent the two methods of precoagulation differ in their clinical effects remains to be determined. The fact that buttermilk, and even acidified milk, has commonly and very extensively been used nearly fat free, while pegnin and chymogen have more frequently been used with whole milk, leaves us little basis for comparison for the fat is too important an item to ignore.

Buttermilk, Acidified or Ripened Milk.—The use of buttermilk had its origin in Holland, where real buttermilk, which was a by-product of the dairy industry, was fed to babies with signal success. The striking results in gastro-intestinal disorders soon led to the placing of buttermilk on a rational basis by de Jager and Taxeiras de Mattos in Holland, and later by Sälge in Germany, where it gained great popularity as Holländische Sauglingsnahrung. As first made, it consisted of one liter of buttermilk made from churned soured cream, to which was added 15 gm. (½ oz.) of flour, and from 60 to 80 gm. (2 to 2½ oz.) of cane sugar. This was boiled, allowing it to well up twice before the sugar was added, and once after the addition. The use of so large an amount of cane sugar especially in dyspeptic infants, for whom the milk was most indicated, was evidently fraught with danger, and led to the use of less sugar in later times. In this country there has always been what would seem a more rational tendency to modify the buttermilk as we would any other milk by dilution and the addition of carbohydrates, as would seem indicated.

For a time emphasis was laid on the use of *real* buttermilk, i.e., made by churning sour cream, and the churn became a part of every well-equipped infant diet kitchen. This, however, soon offended bacteriologic sensibilities, and genuine buttermilk is now rarely used. Commercial buttermilk, which is moreover not buttermilk, but acidified skimmed milk, was objectionable for the same reasons. The word buttermilk is gradually falling into disuse, and in its place we find the term *acidified milk,* either skimmed or whole. This is prepared by boiling the milk, adding to it a culture of lactic acid bacilli of known activity, allowing it to stand at room temperature for 8 to 12 hours, till well clabbered. It is then beaten up gently with an egg beater or a spoon till smooth and liquid. This can then be diluted and modified with carbohydrates, as indicated, as is sweet milk. Of the host of lactic acid cultures on the market, that nearly all claim to be the original Bulgarian Lactic Acid Bacillus, some in liquid and others in capsule or tablet form, some are active but others are quite unreliable. If possible, it is best to get a fresh milk culture of a known active strain, and use this as a "starter," using each subsequent day a part of the acidified milk (1 to 2 oz.) of the preceding day, as a "starter" for the next day. Babies, if not too old, will quickly take to soured milk, and often it is as difficult to return to sweet milk as it was originally to give soured milk. If there is serious objection, especially from the older baby, it is commonly best, at least at first, to add a grain of saccharine to the quart, which can gradually be eliminated, especially if cane sugar is used. It must be borne in mind that fat-free and whole acidified milk are not interchangeable. The brilliant results that are obtainable with buttermilk in gastrointestinal disorders follow the use of fat-free acidified milk. That whole acidified milk can be used as a substitute for fat-free acidified milk to a larger extent than has been generally accepted is probable, but in the more serious cases the fear of fat cannot be eliminated.

"Buttermilk" or fat-free acidified milk is probably the easiest artificial food to digest by the baby. The acid test of digestibility is the foundling home and the hospital ward, and here buttermilk has long ranked in favor as second only to mother's milk. Why this is so is not yet clearly determined. That living, lactic acid producing bacteria have nothing to do with it is evident from the fact that boiling the "buttermilk" as is done in Holland and Germany, and to a large extent here, seems in no way to alter the effect of the food. It seems probable too that relatively few of the organisms escape the stomach in an active form. Digestive processes in the intestine take place in an alkaline medium, and so it is difficult to see how the increased acidity of the food can be useful, whether the milk is boiled or not. Czerny and Keller consider it unproven that the difference between buttermilk and whole milk is not due wholly to the difference in fat content. Experience, however, shows a wide clinical difference between buttermilk and fat-free sweet milk. This view also does not explain the fact that acidified whole milk is easier to digest than whole sweet milk. We are forced to the conclusion that acidified milk has either some as yet undertermined property which makes it easier to digest than sweet milk, or else that difference is wholly or in part due to the fact that when milk is once coagulated and again agitated into a liquid form, it will not re-coagulate in the stomach. If milk precoagulated by rennin would act like acidified milk, this explanation might be accepted. That it does this in part is well known, but to what extent it does so remains to be determined, because of insufficient data now available. Even if it does not, a possible difference in digestibility by the infant of cow's milk casein coagulated by lactic acid, as compared with rennin in the presence of acid, would have to be considered and might be significant.

Pegnin or Chymogen Milk.—Pegnin, a rennin ferment in a milk sugar medium, has had considerable vogue in Germany, and to a very limited extent in this country. Chymogen, a similar product put out by Armour & Company, is used extensively with us in the preparation of Eiweissmilch, and also to some extent for its effect on the casein in simple milk mixtures. A teaspoonful of either preparation is added to a quart of milk that has been boiled for several minutes and allowed to cool to a temperature of 104 degrees, stirred for one minute and allowed to stand at room temperature for about 15 minutes or until a clabbard is formed; it is then beaten or shaken till finely divided and liquid, and then modified in any way that seems indicated by dilution or addition of carbohydrates. Skimmed or whole milk can be used according to indications. Thus treated, milk shows little tendency to coagulate in the bottle, and practically none in the stomach. It would seem worthy of further trial and of more general use than has been given it. Its greater simplicity would commend it above acidified milk, the preparation of which in the home has difficulties and uncertainties, if it could be shown to have a similar action.

Eiweissmilch, Albumen Milk.—This preparation is the logical development of the ideas of Finkelstein concerning the nature and causation of digestive disorders in infancy. These are discussed fully in a subsequent chapter. It has gained a very wide popularity both in Germany and in this country, as a specific remedy in all diarrheas of infancy, due to indigestion, or fermentation. It is prepared as follows: To a liter of *raw* cow's milk is added some form of rennin (pegnin, chymogen, essence of pepsin, *Simon's Labesenz*) and this is kept at a temperature of about 100 degrees for 20 to 30 minutes, until well coagulated. This is then broken up gently, poured into a sterile muslin bag, and in this it is suspended and allowed to drain for one hour without external pressure. The whey is discarded and the remaining curd is gently rubbed three or four times with a pestle or fine spoon through a very fine wire sieve. This should not be done forcibly, and is best accomplished under the gradual addition of a half liter of water. To this mixture a half liter of fat-free acidified milk or buttermilk is added. Various minor modifications have been made, such as the addition of one or two tablespoonfuls of flour, and the immediate addition of a dextrin-maltose preparation (Soxlet's Nährzucker in the original preparation). Under any circumstances a dextrin-maltose preparation should be added as rapidly as possible, up to at least 3 per cent. The whole mixture is brought to a boil and kept there for a short time. During the boiling the mixture must be stirred constantly and actively preferably with an egg beater, otherwise there is danger of the formation of lumps. Eiweissmilch is difficult to prepare except in a diet kitchen. It can be made in the home after careful instruction, but the making of the product itself must be scrupulously watched, especially, if the therapeutic result is not wholly satisfactory. Commonly, if it cannot be procured at some good diet kitchen, it is best to use a dried preparation, such as Hoos' Albumen Milk.

The indication for Eiweissmilch is diarrhea, due to indigestion or fermentation. Its composition, as compared with cow's milk and human milk, is shown at a glance in the following table from Langstein and Meyer:[6]

	Cow's milk, per cent.	Human milk, per cent.	Eiweissmilch, per cent.
Protein	3.00	1.50	2.80 to 3.20
Fat	3.70	3.80	2.00 to 2.50
Sugar	4.50	6.50	1.20 to 1.60
Salts	0.71	0.20	0.45 to 0.50

The action of *Eiweissmilch* is based upon inhibition of fermentation and the promotion of protein decomposition in the intestine. It is accomplished by lessening of the milksugar, and of the whey salts, as well as by the relative increase in the amount of protein and calcium. As a consequence of the changes thus produced in the intestine it becomes possible to add a carbohydrate which less readily undergoes fermentation (a dextrin-maltose preparation), a very important consideration in avoiding a carbohydrate starvation.

The technique of feeding with Eiweissmilch can be varied as one gathers experience, depending upon the exact nature and degree of the disturbance. The beginner will do well to accept the dogmatic directions laid down by Finkelstein and Meyer, until that experience is gained. The Charybdis of starvation lies on one side, the Scylla of overfeeding and return of diarrhea, on the other, and the mariner who sails without a pilot to guide him is very likely to come to grief on one or the other. In the cases where Eiweissmilch is most indicated, in severe marasmus with rapid loss in weight and diarrhea ("Decomposition," Finkelstein); and in severe alimentary intoxications, these dangers are so real that life is seriously threatened by either one.

Langstein and Meyer[6] give the following direction for the treatment of the former of the two conditions: "After 12 hours of a tea diet (a longer time is not necessary, and may be even harmful; a shorter time does not always accomplish the purpose) one begins to give small amounts of Eiweissmilch, to which has been added 3 per cent. of a dextrin-maltose preparation. The initial dose is 300 gm. given in 6 to 10 feedings a day, and continued for three or four days. During this time the primary aggravation of symptoms takes place and generally this is followed by convalescence with cessation of diarrhea. After convalesence is established one proceeds briskly to an increase of food of 50 to 60 gm. every second day until a total quantity of 200 gm. Eiweissmilch per kg. of body-weight daily is reached, but never more than one liter a day. Depending upon the severity of the disturbance and the nature of the stool, one hastens or retards the beginning of an increase in weight. Gain in weight is attained by the addition of carbohydrates up to 5 per cent. of the dextrin-maltose preparation, and in the case of infants over three months old, by the further addition of 1 to 2 per cent. of flour (wheat, or oat). An increase beyond5 per cent. is permitted in those cases in which the gain in weight does not begin with 5 per cent. . . . It is, therefore, permissible in certain cases in which a gain in weight does not occur with less to increase the carbohydrate to 8 or even 10 per cent. The duration of Eiweissmilch feeding should be six weeks in the younger infant and four weeks in the older, but in the older infant one can often get along with less time." The child is then put on the usual milk mixtures. In cases of less severity and in simple dyspepsia with diarrhea, one can proceed much more rapidly. In severe intoxications the initial dosage and progress must be much slower, unless the child has at the same time a severe marasmus (Decomposition) which calls for food at the earliest possible time.

It is perhaps to be regretted that such dogmatic directions should be given, or should seem necessary. The excuse for it lies in the natural solicitude of the originators, in the face of a storm of discussion and protest against this innovation, that the preparation should be used as their extensive experience had led them to believe it should be. It is interesting to note that the strongest condemnation has come from those who have never tried it as directed, and have in some cases even judged it by the result of an individual substitute for the original preparation. In spite of the most extensive discussion, Eiweissmilch has established itself as the nearest substitute for mother's milk now available in the severer forms of fermentative diarrhea, such as those of marasmus, or decomposition (Finkelstein); and of alimentary or gastro-enteric intoxication, or cholera infantum.

3. Peptonized Milk.—While the casein was the *bête noir* of infant feeding, and other methods of adequately modifying it were under veloped and purely empirical, peptonization was considered a valuable weapon in our armamentarium. Peptonization converts the casein into non-coagulable and partly digested albumoses and peptones. Various commercial preparations of pancreatic extract usually put up in so-called "peptonizing tubes" can be used for this purpose according to the directions that accompany them, and the time taken in the process can be varied according to the degree of peptonization desired. Because so many simple methods of modifying the casein are now current, and because predigestion rarely serves any valuable purpose in feeding by mouth, peptonization has fallen into practically complete disuse, except in the very restricted field of rectal feeding.

4. Malt Soup.—Keller's malt soup, a modification of Liebig's original formula, meets a very different indication than the preceding preparations. Its field of usefulness, although at first intended for various digestive disturbances, is now practically restricted to those cases in which the fat of cow's milk leads to an obstinate constipation, with dry, hard, foul stools, together with a failure to gain in weight and other evidences of malnutrition. Malt soup meets this indication by its low fat and milk content and its very high carbohydrate content. It is made in the following manner: Two mixutres are first prepared: No. 1 containing one-third of a liter of cow's milk, into which is stirred 30 gm. (1 oz.) of wheat flour; No. 2 containing two-thirds of a liter of warm water, in which is dissolved, under constant stirring, 100 gm. of Löfflund's Malt Soup Extract. No. 1 and No. 2 are then poured together and slowly boiled for several minutes under constant stirring. Ordinary malt extracts must not be used unless 10 c.c. of an 11 per cent. potassium carbonate is added to each liter of malt soup. This is provided for in Löfflund's, and also a number of our own malt soup extracts, such as Borcherdt's, Maltine, etc., to each ounce of which about 6.79 gr. of potassium carbonate has been added. A quart of malt soup so prepared contains about 700 to 800 calories.

This mixture contains a minimum of fat, protein and salts, except potassium salts, and a maximum of carbohydrates made up of four varieties, starch, dextrins, maltose and milksugar. It naturally favors fermentative processes and reduces putrefaction to a minimum. For this reason it must be given cautiously, especially to young infants, and is best not used before the fourth month for fear of producing diarrhea. Later in the second half year, the amount of protein is not enough to meet the infant's needs. These objections can be met, on the one hand, by using less malt extract, and on the other hand, by giving more milk or adding dried casein (plasmon, nutrose, Laroson, sanatogen, or pure dried casein), or coagulated and finely sieved skimmed milk curd. Within these limits and to meet this indication Malt Soup is an excellent preparation to be used, however, only for a limited time. Both the low milk content and, possibly, the richness in alkalies,[24] make it weak in antiscorbutic properties.

At one time, very popular, the use of Malt Soup is now very limited. This is due mainly to the fact that practically the same result can be produced in the first half year by lowering the milk, possibly partly skimming it, and adding a large amount of dextrin-maltose and a smaller amount of flour; and in the second half year, by restricting the milk, and feeding freely cereals, zwieback, toast, crackers and vegetables.

THE PRACTICAL PREPARATION OF AN INFANT'S FOOD, AND THE CARE OF BOTTLES, NIPPLES, ETC.

The actual preparing of a baby's food is a simple process that requires no special apparatus other than that found in the ordinary kitchen, except that there should be at hand some accurate measure graduated to ounces, or to c.c., such as a glass graduate. Ordinary clean sauce pans are ample for all mixing and boiling purposes. Scales for weighing ingredients are wholly unnecessary as the amount of sugar, flour, etc. can be expressed accurately enough in terms of level tea- or tablespoonfuls. The following table gives the approximate number of level teaspoonfuls or tablespoonfuls, leveled (and not packed) with a knife, of the ordinary ingredients, that correspond to 1 oz. by weight:

One ounce by weight of cane sugar = 6 to 7 teaspoonfuls.
One ounce by weight of milksugar = 10 to 11 teaspoonfuls.
One ounce by weight of Dextri-Maltose = 14 to 15 teaspoonfuls.
One ounce by weight of flours (barley, wheat, etc.) = 15 teaspoonfuls.
One ounce by weight of rolled oats = 6 tablespoonfuls.
One ounce by weight of pearl barley = 2½ tablespoonfuls.
One ounce by weight of dried milk = 8 tablespoonfuls.

Teaspoons are more constant in size than tablespoons and should be used except for such preparations as the last three which cannot be leveled accurately in a teaspoon.

The following table gives the approximate weight and measure equivalents of the more commonly used ingredients:

One ounce by weight of cane sugar = 1− oz. by measure.
One ounce by weight of milksugar = 1.25+ oz. by measure.
One ounce by weight of dextri-maltose = 1.75 oz. by measure.
One ounce by weight of flours = 1.75 oz. by measure.
One ounce by weight of dried milk = 3.50 oz. by measure.
One ounce by weight of rolled oats = 2.30 oz. by measure.
One ounce by weight of pearl barley = 1.10 oz. by measure.

The **bottles** should have relatively wide mouths and should be so shaped that all parts are easily accessible to a bottle washer. There should be no sharp interior angles or corners. The large, heavy, straight, cylindrical bottles with a mouth of the same caliber as the bottle would seem to offer no special advantage as they are really not more easily cleaned. They are moreover very cumbersome to the baby, and take up a great deal of room in the ice box. The nipple, too, is commonly not soft or of a specially desirable shape. It is very convenient to have a bottle rack to contain all of the day's bottles, as the best bottles are rounded and do not stand well alone unless so fixed by a support.

The **nipples** should be small, soft, relatively non-collapsible and invertible for washing purposes. In my experience these requirements are best met by the so-called "anticolic" nipple with a spherical end with three holes. The medium size is the one commonly most serviceable, but for the small baby the small size is often preferable. The holes should be of such size that when the bottle is turned upside down the food will run out in drops, not in a stream. If the hole is too large the nipple should be discarded, if too small it can be enlarged to any desired size by momentarily plunging a red hot small needle into the end of the nipple. For thicker foods, such as Eiweissmilch, the holes should be larger than for the usual mixtures.

Both bottles and nipples should be washed, not scalded, immediately after being used and then set aside. A new nipple should be used each time and it is best to have a few more nipples than bottles. All of the nipples should be washed in soap and water, and then boiled for a few minutes, before being used again. They are then placed *dry* in a cup with a saucer inverted over it and used as needed. They will, thus kept, last longer and be less soft than if kept in some solution and are at least as sterile. The bottles should be washed thoroughly with a bottle washer in hot water and soap daily and then thoroughly rinsed and dried. It is customary to boil them for 5 or 10 minutes, though it can at least be seriously doubted whether this has any practical advantages over thorough cleanliness as outlined above. When confronted by the modern young mother it is expedient however not to omit this, even though it is very wasteful of both time and bottles.

The **food** should be made up *for the whole* 24 *hours* at one time, preferably in the morning so that the milk can be consumed as fresh as possible. If the milk is to be boiled or pasteurized, this can be done first. If it is boiled this is best done in an open sauce pan, on an asbestos mat, under constant stirring. The number of ounces of milk called for in the formula should be supplied by the number of ounces before boiling, not after boiling, as the amount is reduced by the loss of water through evaporation. If the milk is to be pasteurized it should be heated to 150 to 155 degrees in a double boiler and kept at that temperature for 30 minutes and then cooled rapidly. The milk and diluent are then mixed and the sugar added. If a starchy ingredient, such as barley flour, or flour ball, or oatmeal, is used in the mixture this must first be boiled in the water alone before the milk is added.

The **cereal waters** so commonly used are prepared separately and then added to the milk. *Barley water*, the most commonly used, can be made either from the barley flours, such as Robinson's, or Johnson's, or from pearl barley. The latter is commonly used in diet kitchens, the former in the home because it is apparently equally serviceable and less time and fuel consuming. *Rice, wheat, or oatmeal water* can be made in the same way either from the flour or from the grains, in the case of oats from the rolled or Quaker oats. When the flour is used in any of these one rounded tablespoonful is added to one pint of water and boiled 20 minutes in a sauce pan under constant stirring, or for 45 minutes to an hour in a double boiler. Water is added so that at the end of the time there is a pint of the cereal water, or one can begin with a larger amount of water and boil it down to one pint. This is then strained through coarse muslin or a fine sieve and a pinch of salt is added. If the grains are used one full tablespoonful is added to each pint of water and this is boiled for three to five hours, keeping the water at the original amount. This is then strained and salted as before. It is customary to soak the grains in water for a few hours, or over night, before boiling. The water in which they are soaked is not used. I have never been able to see that there is any practical difference between the cereal waters prepared from the flours and those prepared from the grains. Barley water made from the grains is commonly darker, browner in color. There is no conclusive evidence that there is any physiological difference in the cereal waters from different grains, such as, for example, in the effect on the bowels, or on milk coagulation, or in digestibility. Cereal waters must be kept rigidly on ice, and especially if enough is made at one time to last two days, as they ferment readily. They should be made fresh daily.

Oatmeal, barley, wheat, or rice jelly for spoon food can be prepared as are the cereal waters, using however twice the amount of cereal.

Cereals can be cooked as for the adult table provided they are cooked thoroughly. Oatmeal especially should be cooked several hours, preferably over night in a fireless cooker. It is not necessary to strain them and toward the latter part of the first year it seems to me even objectionable to do so.

Broths can be made from beef, mutton, veal, or chicken. While the laity and many physicians attribute peculiar physiological attributes to one or the other of these, notably mutton broth, as compared with the others, there is, to my knowledge, no tangible evidence that such differences exist. Broths for babies are commonly made from meat, not from a bone. I have for many years used the following formula from Holt as a routine: One pound of round steak cut into small pieces, one pint of water; cook on a slow fire for two hours down to 8 oz. Strain, cool and skim off the fat. If desired this can be thickened with any flour added before boiling.

Vegetable soup can be made according to the directions for making broth. A carrot, a small potato, a small handful of spinach, a stalk or two of celery, and possibly a little onion, all finely cut up, can be added to the mixture at the beginning and boiled with it. The meat is then separated from the rest and the broth and vegetables passed through a fine sieve.

Beef-juice is best made as follows: A piece of round steak is lightly broiled, or seared over in a hot skillet, on both sides, and the juice pressed out by a meat press, or lemon squeezer. Two or three ounces can commonly be obtained from 1 lb. of steak. This is seasoned with salt and given cold or warm, separately or in the bottle.

A more *economical method of preparing beef-juice*, less palatable and rich in extractives, however, is the following: To a pound of finely chopped round steak add 8 oz. of water and let this stand on ice for six to eight hours in a covered mason jar. The juice is then squeezed through coarse muslin and seasoned. This will yield over 8 oz. of beef-juice.

The following table compiled from Holt gives the relative composition of beef-juice prepared by these two methods and of broth made as above:

	Beef-juice from 1 lb. of steak, warm process; quantity 2½ oz.	Beef-juice from 1 lb. of steak, cold process; quantity 8½ oz.	Beef broth, made from 1 lb. of steak; quantity 8 oz.
Proteins	2.90	3.00	1.02
Fat	0.60	0.00	0.00
Extractives	3.40	1.90	1.82
Salts	0.20	0.20	0.88
Water	92.90	94.90	96.28
Total	100.00	100.00	100.00

There is very little, if any, clinical evidence that beef juice has any advantages over broth either in the matter of food value or in the amount and nature of the extractives. It is always customary to give a larger amount of broth than of beef-juice and this equalizes any deficiency in the former. The chemical analyses above likewise show no differences of value. For these reasons beef-juice has largely been replaced by broths, in my own practice wholly so. The fact that vegetables can be combined with the latter is a further advantage.

Scraped beef is the form in which meat is commonly first given. It is prepared by searing over in a hot skillet the surface of a piece of round steak and then scraping that side with a spoon or dull knife. It can also be prepared by running a piece of round steak through a fine meat grinder, making a pattie of the ground meat and broiling this lightly. The former method has the advantage of eliminating the fiber of the meat. By either method the meat is nearly raw, but when seasoned with salt is rarely refused by the baby. Scraped beef offers no practical advantage over finely cut up broiled steak, or lamb chop, except when given, for some therapeutic reason, to a younger baby at an age when meat is commonly not given as yet.

FEEDING IN ACUTE ILLNESS, OTHER THAN GASTRO-INTESTINAL (PARENTERAL AFFECTIONS)

This subject would hardly seem to warrant the detailed discussion usually given it. Nature commonly provides adequately for this condition by taking away the child's appetite to a safe degree. Temporary complete withdrawal of food can serve no useful purpose in other than gastrointestinal disorders. It has long been my own custom to let a child eat all of the simpler things that he has been accustomed to during any illness that is not accompanied by diarrhea, and I have seen no reason to regret that course. Even in cases that vomit and have no diarrhea, therefore in the infant no serious gastro-intestinal disorder, the vomiting is so often reflex that it makes little difference whether a small amount of food is given or not. In short febrile attacks the degree of appetite varies strikingly. Feeding should be here never forced, but if the child wants food it can commonly utilize it without harm, in such amounts as it will take. In all febrile cases, to make a distinction between milk as a liquid or non-solid food, and cereals, toast, puddings, potato, etc., as solid foods is no longer tenable, unless the interpretation of that distinction is the reverse of that usually accepted. Commonly the sick child, especially if it has a fever, prefers a liquid food because it satisfies thirst at the same time, though often it makes a sharp distinction between water and any liquid food. The desire for water should here be satisfied to the degree desired. There is, of course, no longer the objection to cold water that once existed. The same is true of food, and ice cream is often accepted when nothing else is. Even if a child will take almost no food during a short acute illness, such as lobar pneumonia, this need not concern us over-much. Water must, however, be supplied in sufficient amounts and in stuporous or semi-conscious individuals, even if it must be given with the stomach tube, or by rectum, or by hypodermoclysis. With the beginning of convalescence and the slow return of appetite, there is often a craving for special things. If this is at all within reason it should always be granted, for it often seems to stimulate the appetite for other things.

MIXED FEEDING. ALLAITEMENTE MIXTE

By mixed feeding is meant the combination of artificial feeding with breast feeding. It is indeed a strange fact that today we still find the idea prevalent, both among laymen and medical men, that cow's milk and mother's milk do not go well together; that when the mother does not have enough milk, the baby must be weaned and put on a bottle. Nothing could be further from the truth. Mixed feeding is not only feasible, but a most happy combination, as pediatrists have long known. In fact, properly conducted mixed feeding is commonly quite as successful as breast feeding alone. As long as the mother has 5 or 6 oz. of milk a day, the progress of the case resembles more that of breast feeding than that of artificial feeding. The same is true of the artificially fed baby in the first few months of life, that gets a feeding or two of breast milk from some source. So true is all this that the question of when and how to wean a baby has been quite replaced by the question of how much artificial feeding it is necessary to add to the mother's supply to insure normal progress.

Mixed feeding can consist either of *substituting* one or more bottle feedings for an equal number of breast feedings, or else of complementing each breast feeding with a bottle, to the extent that seems indicated. Which of these two procedures to follow depends upon the age of the baby and the amount of milk given by the mother. If the baby is under three months of age supplemental feeding is the rule. After the fourth month it is nearly always best to *substitute*, unless the mother has so little milk that she does not have enough for two good breast feedings a day, and it becomes a matter of indifference whether nursing is continued or not. If the mother has an abundance of milk and for some reason it is desirable to relieve her of one feeding a day, then the bottle is of course substituted for one breast feeding at any age. If she has nearly enough then, too, a substitute bottle can be given at any age. If she has so little that the baby is not gaining, or is actually losing in weight, then the question of substitute or supplemental feeding must usually depend on the age of the baby.

Supplemental feeding is the method of choice in the first three months, for several reasons. There is at this age no danger whatever that the baby will give up the breast, because of the knowledge that it will be succeeded by the more desirable bottle. Such intelligence seems to come only with the later months, when it becomes a real source of danger of self-weaning. This is especially true if the mother has relatively little milk and if it is harder to get from the breast than from an easy-flowing artificial nipple. On the other hand, there is again little danger of self-weaning after the seventh or eighth month if the breast milk has been, and still is, abundant, and the baby has been nursed exclusively. Even if it has been taught to use the bottle, the breast habit has become so fixed that it remains preferable to the bottle, or at least equally acceptable. Supplemental feeding is also to be preferred at this time because one wants to be sure that the baby gets enough at each feeding, which might not be the case at the times when the breast alone is given. It also insures, at a time when it is most needed, the only real stimulation to milk secretion that we know of—the regular, fairly frequent and thorough emptying of the breast.

It is best to prepare as many bottles as there are feedings a day, giving one after the breast at each feeding, alternating the breasts at successive feedings, and giving only one each time. After the longer period at night, the breasts will often contain enough in the morning for one or two feedings of the breast alone, and the bottle need then be given only after the subsequent breast feedings. Often it is well to give increasing amounts after each successive nursing, for it is the rule that the breasts have increasingly less milk as the day advances. In giving an artificial food for the first time in supplemental feeding, one naturally follows the same general course as in artificial feeding alone, except that one can begin with a stronger mixture and can increase it more rapidly. This is true both because breast milk in some way disarms cow's milk and also because the latter is still further diluted by becoming mixed with the breast milk in the stomach.

The *amount* of bottle feeding to give after each nursing depends on how much the baby is getting from the breast, and upon how much it is gaining or losing. To the experienced clinician the mother's story and the general condition of the baby, the weight, and the number and nature of the stools, and an examination of the breasts is all that is necessary. Until that experience is acquired it is well to get an idea of how much the baby is getting from the breast in the 24 hours by weighing him occasionally *at different times of the day* before and after nursing. The estimated daily amount so obtained can then be compared with his fairly accurately known requirements, expressed in ounces of milk per pound of baby, daily; or relative amount of milk to weight of baby; or in calories per pound, or kilogram. In general, a baby requires approximately 2 oz. of breast milk to the pound of baby, daily; or about one-seventh of its weight daily; or about 45 calories per pound, or 100 calories per kg. of body-weight daily. All of these figures are a little low for the beginning, and a little high for the end of the first half year. As an example, if a baby of four or five months of age, weighed 14 lb. and was estimated to get 30 oz. of milk a day, this would be about right, because it would be getting about 2 oz. of milk per pound; about one-seventh of its weight of milk daily; and about 45 calories per pound, or 100 calories per kg. of body-weight daily. Used as a guide, not as an advance measure of what a baby must get, or as a complete substitute for clinical observation, one or the other of these procedures is, to the beginner, of great value in supplemental feeding.

Weighing the baby before and after each and every feeding, and then supplementing to the calculated desirable amount to give at each feeding, as seems to be practiced now to an increasingly larger degree, is not only not necessary, but seems to me one of those meddlesome hyper-refinements that crop out every so often in all branches of medicine. Such a procedure does not take into account maternal psychology, that in the nursing mother is often as important as the baby itself. Checking up from time to time the amount of milk obtained from the breast does not make the mother nervous if it is properly done, and is a valuable guide in supplemental feeding, but the progress of the baby and all the attendant circumstances make even this quite unnecessary after a little experience. In a general way one can safely put a little more in each bottle than one thinks the baby commonly needs, and let it take as much of this amount as it wants to. The breast milk will see to it that no harm is done from a little overfeeding, and both overfeeding and underfeeding can amply be controlled by the weekly weight curve.

After the third or fourth month **substitute feeding** must commonly replace supplemental feeding. The danger of self-weaning from the breast, that is minimal during the first few months, now becomes very real. Only if the mother has very little milk and discontinuing the breast is only a matter of a very short time, and especially if she does not have enough for even two adequate breast feedings a day, is it, as a rule, permissible at this time to give the bottle after the breast. In *substitute* feeding one replaces as many breast feedings by bottle feedings as seems indicated, each bottle holding as much food and of such strength as one would give to a baby that was to be fed artificially alone for the first time. It is here necessary to proceed a little more cautiously than in supplemental feeding because the cow's milk is not diluted by breast milk and has to be given in larger amounts at one time. If the baby has been well nourished up to the present time, it is well to begin with one bottle, preferably the third feeding in the day, for obvious reasons. When a second bottle becomes necessary it can be identical with the former in size and strength. The two bottles are best so spaced that the breast feedings come first in the morning and, alternating at successive feedings with the bottle, constitute also the last feeding at night. It is now best to give both breasts at each breast feeding. When three bottles are given the breasts should be given at 6 :00 a.m. and 6 :00 p.m. (or 10 :00 p.m.), and the bottles at 10 :00 a.m., 2 :00 p.m. and either 6 :00 p.m. or 10 :00 p.m., depending on the preference of the mother. When only two feedings a day can be given from the breast, weaning is not far off. When these two feedings become inadequate, one can either supplement them by a bottle, or else resume supplemental feeding at each nursing, fully expecting the baby to refuse the breast altogether in the course of a short time. In this way the transition to artificial feeding alone is as gradual as possible, as it always should be under normal conditions. Weaning is thus not a kind of rite to be performed at a certain day or a certain age; it consists rather of giving the baby enough other food and no more, so that he will progress normally and continuously; the artificial feedings always so planned that the breasts will yield the largest amount obtainable from them.

Spoon food, such as cereals, broth, zwieback, orange juice, etc., can be given at the same age and in the same manner as in the artificially fed baby, and are best given with the bottle feedings.

The *first two weeks of life*, and especially the first week, deserve more attention than is commonly given them, either in practice or in our text books. At no time in life should the baby's feeding be watched more carefully; at no time are the results more fateful, and yet I believe it is safe to say at no time does the baby get less careful attention than during this critical period. This is all the more to be regretted because at no time is the treatment more simple, and clear cut, and the results that come from careful observation and timely intervention in the matter of mixed feeding more gratifying. Midwifery at first included this part of pediatrics and later the two went together more or less naturally in practice. Obstetrics has become a highly specialized branch of surgery, with the interest centered on the mother rather than on the baby, and this interest only naturally does not change after the delivery. In a very large proportion of babies, the question of mixed feeding presents itself in the first two weeks of life, a problem that always demands the most tactful management, and never more so than at this most delicate period. In spite of these facts that are self-evident, the baby is only too commonly left wholly to the care of a nurse, or interne, or to some inflexible routine, until a condition arises that is often alarming and that always makes feeding more difficult than if it had not occurred.

This confusion is increased by the fact that babies normally, for some unaccountable reason, get very little food from the mother for a variable number of days after birth. Waiting for the mother's milk to come in sufficient amounts is easily prolonged for too long a time, during which time the baby loses more than the physiological amount of one-eleventh to one-tenth of its weight. Starvation, or under-nourishment, is always in itself harmful. Here it serves no useful purpose, as it does in digestive disturbances; indeed, it may lead to serious and even irreparable harm. In spite of this self-evident fact, babies are allowed to lose abnormally much and far beyond the first two or three days. Such useless starvation commonly leads to an inanition fever and an accompanying apathy that often taxes all of one's ingenuity in getting the baby back to a condition in which he will even nurse again. Only too often the mother loses the milk because of worry, and because the baby in his weakened condition will no longer nurse, and so deprives her breasts of the only real stimulation to milk secretion. The baby finally finds himself cheated of his birthright, and starts the hazardous journey with at best a poor substitute. Perhaps even more often, the mother has only enough milk so that the baby shows neither gain nor loss on the scales. If he is "holding his own" active treatment is commonly delayed, in spite of the fact that "holding his own" after the third day means a real loss of about an ounce a day, which he should have gained. The baby that in the first two weeks of life is "holding his own" is not only losing rapidly, he commonly gets less vigorous, becomes apathetic and nurses less well. The mother deprived of that stimulation and also of mental repose, yields less milk, the baby's condition gets worse, and we have established a vicious circle that leads to disaster if it is not broken.

The remedy is of the simplest possible, and permits of dogmatic statement. For all practical purposes it can be accepted as a fact, that knows few exceptions, that *the baby that is breast fed and does not gain during the first two weeks of life, that is not vomiting too much to make a gain possible, and that has no diarrhea with the resulting loss of food, is not getting enough to eat, for one reason or another.* If then a baby continues to lose beyond what may be considered a physiological amount and length of time, or remains stationary in weight, or has the slightest fever, and there is no immediate probability of his getting enough from his mother, he should at once have supplemental feedings from the bottle of the same size and strength as one would give to any newborn baby. If human milk is obtainable the problem is greatly simplified. The bottle should of course always be preceded by the breast. Almost without exception, the result is wholly gratifying; the baby takes on new life and vigor and nurses better. If the mother has any milk at all the stimulation of the breasts that comes from vigorous nursing, and the peace of mind that comes from knowing that her baby is progressing favorably will assure the largest amount of milk that it is possible to obtain from her. *Any therapy at this stage that fails to give proper weight to the mother's state of mind is less than half effective.* With proper maneuvering the baby can often ultimately be left wholly on the breast, while if mixed feeding had not been carried out in the right manner the baby would have been wholly deprived of the breast. If breast feeding alone is not possible ultimately, mixed feeding can commonly be continued for weeks and months with almost equal success. The fact can hardly be emphasized to strongly that at no time while on the breast alone should a baby fail to gain a normal amount and that continuously; that under-nourishment is never negligible, and that in the first two weeks of life it is attended by serious dangers.

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CHAPTER XXIV

DIABETES MELLITUS

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Definition.—"Definitions of Diabetes are unsatisfactory, but it is safe to say that diabetes is a disease in which the normal utilization of carbohydrate is impaired, in consequence of which glucose is excreted in the urine." (Joslin).[1] Glucose appears in the urine in other conditions than in diabetes mellitus. This will be more fully discussed later under diagnosis. Lusk[2] distinguishes between "glycosurias where the *sugar-holding* capacity of the organs has been diminished or overstrained, and the glycosuria of *diabetes* in which the *sugar burning* capacity has been affected."

History.—According to Epstein,[3] definite mention of most of the cardinal symptoms of diabetes is found in the literature of the Chinese of some six centuries B.C. The Bible and the Talmud, however, make no mention of diabetes, but the Jews of those days apparently suffered frequently from boils, carbuncles and bulimia. The name "diabetes" was first given to the disease by Aretaeus. Both Galen and Celsus spoke of diabetes, although their conception of the disease was entirely different from present day ideas. Foster,[4] also, states that the saccharine nature of diabetic urine had been known to Hindu physicians from remote times. Dobson, in the eighteenth century, was the first to demonstrate sugar in the urine, but sugar in the blood was not demonstrated until 1836 by Ambrosian. In 1826, Prout[5] stated in a very clear essay on the disease that no sugar is in the blood even in diabetes. Claude Bernard[6] in the middle of the nineteenth century published his experiments on the relation of the liver to sugar formation, which opened the way for the modern physiological studies on the disease.

Throughout the history of diabetes, improvement in therapy has advanced ahead of knowledge of the scientific basis of the disease. Rollo[7] in 1796 was probably the first to call attention to the dangers of carbohydrate diet. In 1826 Prout, (*loc. cit.*) laid down a scientific basis for treatment. From this time until the latter half of the nineteenth century no considerable advance in treatment is recorded. In 1874 Kussmaul[8] described diabetic coma. Magnus-Levy,[9] Naunyn[10] and von Noorden[11] stand out as prominent contributors to the modern therapy of the disease. Minkowski and von Mering[12] proved the relation of the disease in experimental animals to pancreatic disturbances. This work has recently been confirmed and extended by Allen.[13] In 1922 what seems to be the final link in the evidence proving the pancreatic nature of the disease was forged by Macleod[14] and his co-workers at Toronto who isolated a pancreatic extract, *Insulin*, capable of reducing blood and urine sugar in normals and diabetics.

ETIOLOGY

Heredity.—The absolute cause of diabetes is unknown at present, but in the discussion of the disease there are several etiological factors of more or less importance, one of which is heredity. Especially in children is hereditary influence to be sought, since certain observers have maintained that the only mild cases of diabetes in children that they have ever seen have been those in which there was a definite hereditary taint. Among our cases of diabetes in children an hereditary influence has not been found. Abt[15] says that in families in which several generations have been afflicted with diabetes, the patients are younger in succeeding generations.

It is no uncommon experience to find large diabetic families. In our experience, this is especially true of diabetes in the adult, but it is a rather striking fact that most of the children that we have seen have been exceptions to this rule. We have seen no mild cases of the disease in children. Joslin's[16] cases of hereditary diabetes "have frequently been mild, and this has so commonly been the case, that I always have looked upon heredity as a favorable omen. Several cases in young people have been unusually favorable. All cases which have come to my attention of youthful patients with diabetes living for such long periods of time, have been hereditary." Joslin (*loc. cit.*) cites four cases with a hereditary history with an unusually long duration, as can be seen from the following table:

Case No.	Age at onset, years	Hereditary history	Outcome	Duration	
				Years	Months
203	7	Mother	Alive	10	1
295	14	Father, also brother	Coma	12	
304	13	Grandfather, also two brothers	Coma	9	2
310	17	Mother, also brother and sister	Coma	21	

Lion and Moreau[17] report 23 cases out of 100 in children which showed a familial occurrence of the disease. Langaker[18] reports the incidence of the disease in four children of a family of eight, all of whom died within four years. Three of the four children were less than five years old at the time of death. Five years later a fifth child of the same family died at seven years. Lion and Moreau (*loc. cit.*) collected 30 incidences of juvenile diabetes with a hereditary history.

The relation of childbirth to maternal diabetes is interesting. Diabetes in the mother is frequently disastrous to the fetus. Premature labor, with dead or macerated fetus, is common. Naunyn[19] reports a diabetic woman who had three successful pregnancies, each of which resulted in abortion at the fourth or fifth month. Offergeld,[20] reporting a series of 57 diabetic women, showed that 51 per cent. of the children were stillborn, 11 per cent. died soon after birth, and 7 per cent. died in infancy from hydrocephalus or diabetes, making a total mortality of 66 per cent. In 25 cases in which the children died, 17 of the mothers died in coma. Naunyn (*loc. cit.*) says that there is no well authenticated case on record where a diabetic mother gave birth to a diabetic child.

Race.—Diabetes is commonly considered to be more frequent among certain races than others. Foster[21] says, "It has long been a tradition that certain races are more susceptible than others to this disease, and the Jews are commonly cited as an example. Of 400 cases in Frerich's[22] series, 102 were Jews. The susceptibility of the Hebrews is best attested by the careful survey of Frankfurt by Wallach,[23] who found that the proportion of deaths from diabetes to the total deaths from all other causes was six times greater among the Jews than among the other inhabitants." Williamson[24] has shown that the mortality from diabetes among the Jews was greater at all ages. Of 1487 diabetic patients treated by von Noorden,[25] 31.5 per cent. were Jews, while Joslin[26] has treated 102 Jewish patients. Morrison[27] (1916) analyzing the mortality statistics from diabetes in Boston from 1895 to 1913 finds some most interesting racial characteristics. During the period embraced by the study there were 1775 deaths from diabetes mellitus, of which 127 were among Jews. The total Jewish deaths during this period were 6936, and total non-Jewish deaths were 222,532. The figures as expressed in percentage of deaths from diabetes to total deaths are for Jews 0.018; non-Jews 0.007; German descent 0.014; English 0.013; American 0.012; Irish or Canadian 0.009. Abt (*loc. cit.*) says, "It would seem more than coincidence that I have seen seven to eight non-Jewish children with diabetes to one Jewish child." Epstein (*loc. cit.*) finding no reference to diabetes in the early Hebrew literature, casts some doubt on the accepted supposition that diabetes amongst the Jews is unusually common.

Social Status.—Undoubtedly social status plays a rôle in diabetes in the adult, and most available statistics show that the well-to-do are attacked by the disease in greater proportion than the poor. It is, however, difficult to extract from the many possible factors (better food, more sedentary habits, greater nervous tension) that particular one which might be important. In the Michael Reese Hospital diabetes is much more common in the private rooms than in the wards. No statistics as to social status of youthful diabetics are available:

in our experience it seems to have played no rôle, as our cases are divided equally among rich and poor.

Age and Sex.—Diabetes may be found at all ages and is more common with males than females. The following table quoted by Foster[28] from Mortality Statistics, Bureau Census, 1909, illustrates the incidence of the disease in regard to age and sex.

Years	Male	Female
5	38	45
5– 9	51	65
10–14	68	107
15–19	113	71
20–29	205	161
30–39	232	187

Wegeli[29] collected 108 cases of diabetes under 16 years of age and was able to give the age of occurrence in 102:

Age	Male	Female	Not Given	Total
Under 1 year................	2	..	1	3
1 to 5 years................	12	8	6	26
5 to 10 years................	11	17	3	31
10 to 16 years................	19	22	1	42
	—	—	—	—
	44	47	11	102

It is to be noted that in Wegeli's table boys are more frequently affected up to five years, while in the census report girls are slightly more affected.

Joslin's[30] cases shows a proportion of 60 men to 40 women; while of 85 cases in children under 15 years 45 (53 per cent.) were males, 40 (47 per cent.) females.[31]

Knox[32] in 1913 collecting statistics on the incidence of the disease in children reports many interesting data. Of 6496 fatal cases in Wales and England in 1865 to 1870 only eight cases were under one year. Knox's own case was nine months old. After analyzing many of the cases reported in the literature (1852–1913) he finds 16 under one year of age that were probably cases of real diabetes as distinct from 11 others reported as diabetes but which are doubtful.

Probably the youngest case of diabetes (?) ever known is that of Morrison (*loc. cit.*) from the Boston Mortality Statistics of 1895 to 1913, namely, a newborn infant taken from a diabetic mother who also succumbed.

Trauma.—Trauma has for many years been considered one of the causes of diabetes in adults and children. Abt and I[33] were fortunate enough to be able to study thoroughly two cases of diabetes in children which definitely followed injury. In one of these cases the injury was to the head, in the other to the abdomen. The sugar was not present before the accident, so that there seemed to be a definite etiological relationship between the accident and the onset of the disease. The disease which does follow trauma differs in no respect from true diabetes. In both our cases, complete clinical and metabolic studies were possible, and in both, we were able definitely to prove that we were dealing with true and severe diabetes mellitus.

Hoeniger[34] studying a series of obstetric cases showed that an ephemeral traumatic glycosuria could be discovered by examining the urine of newborn babies after operative obstetric interference. This glycosuria is not found after other labors, even when prolonged, and according to Hoeniger's interpretation is due to the trauma. Foster[35] reports a case of diabetes in a child four years of age whose symptoms began within 24 hours after the boy had been in a railroad accident but who had not suffered any injury. Diabetes in children has followed severe trauma not only to the head but to the muscles and abdomen as well. Higgins and Ogden[36] found glycosuria in 20 out of 212 head injuries admitted to the Boston City Hospital. Of Naunyn's (*loc. cit.*) 50 cases of traumatic diabetes half followed head injuries and half followed injuries to the other parts of the body. Abt (*loc. cit.*) reports a case in a child of four whose playmate let a brick fall upon his head from a distance of 4 ft. Not even a scalp wound was produced by the injury, but within a week the patient showed marked polyuria, voracious appetite and loss of strength. The patient came for observation seven weeks after the accident, and urinary examination at that time showed 2.8 per cent. sugar.

Nervous Influences.—It is very probable that the effect of trauma on the onset of diabetes is closely related to the central nervous system. Practically all authorities agree that in the production of diabetes the nervous system plays an important role. Ever since the work of Claude Bernard (*loc. cit.*) on the production of glycosuria by puncture of the fourth ventricle, much interest has attached to the relationship of the nervous system to diabetes. The most recent theory of the disease advanced by Allen,[37] is that diabetes is primarily a functional disease of the nervous system, a conception previously suggested by Naunyn (*loc. cit.*). Whether that be true or not, it seems certain that glycosuria can be caused not only by organic disease of the nervous system but also by functional overstrain or even emotional excess. Foster (*loc. cit.*) reports a case of a child who was severely frightened but uninjured in a runaway accident, and shortly afterwards developed severe diabetes. Folin[38] reports studies from a psychopathic hospital in which he showed very clearly the influence of excitement in producing glycosuria among the insane. Folin, Dennis, Smillie[39] also showed that 18 per cent. of a certain group of second year medical students passed a small amount of sugar immediately after an examination, while 17 per cent. of second year women medical students at Simmons college passed sugar in the urine after an examination. Cannon[40] did an interesting experiment to prove that the appearance of sugar in the urine may arise purely from emotional excitement. Three cats were bound for varying periods up to four hours, but these animals remained calm and passive in their confinement. They showed no sugar in the urine after this test. However, when they were frightened by the barks of a little dog and showed much evidence of excitement, all three cats exhibited glycosuria.

Further evidence is not lacking of a close relationship between the central nervous system and diabetes. Traumatic diabetes or traumatic glycosuria usually follows injury to the head or central nervous system. Examining urine within 24 hours after injury, Foster[41] found sugar in 14 cases (70 per cent.) out of a total of 20 cases of skull fracture admitted to New York Hospital. It is not uncommon in hospital practice to find glycosuria in various traumatic or other pathologic disturbances of the brain, and frequently considerable diagnostic difficulty arises from this association. The studies of Cushing[42] and his co-workers seem to show a very close relationship between the function of the pituitary body and the carbohydrate regulating mechanism. The removal of the posterior lobe of the hypophysis in dogs markedly increased carbohydrate tolerance, whereas injections of extract of the posterior lobe caused glycosuria. Also, many cases of acromegaly show either an actual glycosuria or a diminished sugar tolerance.

Von Noorden,[43] while not believing in the doctrine of a "neurogenous diabetes," urges the necessity of realizing the neurasthenic factor in treatment especially of the milder cases. Naunyn[44] gives an excellent discussion of the relationship between glycosuria, diabetes and injury and disease of the nervous system. Reviewing in detail the literature up to date and his own experience, he believed a direct relation existed between "the neuropathic disposition" and diabetes.

Ductless Glands.—The knowledge that certain diseases of the ductless glands, such as hyperthyroidism or hyperpituitarism are frequently associated with either glycosuria or marked diminution of carbohydrate tolerance, has led some investigators to the belief that the ductless glands play an important role in the etiology of the disease. The von Noorden school[45] has done extensive research in the attempt to prove an inter-relationship of pressor and depressor effects of the thyroid, hypophysis and adrenal on the pancreas. Cushing's (*loc. cit.*) work on the sympathetic ganglia should, perhaps, be noted more under the influence of the nervous system than the ductless glands. There can be no question that these glands do play a role in carbohydrate metabolism, but it is very doubtful if they produce true diabetes.

In children the incidence of disturbance of the ductless glands, especially in relation to glycosuria, is infrequent. One of my cases of renal glycosuria showed some symptoms of hypophyseal disturbances. Also, the highest sugar tolerance and lowest blood sugar I have ever seen was in the case of a patient with Froehlich's syndrome.

Siegmund[46] reports that thyroid deficiency in children is often accompanied by pathological sugar hunger and that such children are able to take large quantities of sugar for long periods without glycosuria. The sugar seems to act beneficially on the thyroid deficiency, and thyroid feeding might cure the sugar hunger within two days. Gordon[47] reports two cases, apparently showing a relation between thyroid and diabetes. The patients were brothers, age three and four and one-half years. The younger child gave full evidence of myxedema with retarded intellect and considerable indifference to his surroundings. The respirations were slow, pulse 60; hair was coarse and the skin thick and edematous. The urine had a high specific gravity, and a few drops promptly reduced Fehling's solution. The child had symptoms of diabetes, such as frequent and abundant micturition and increased thirst. The older child had been sick for two years with marked symptoms of cretinism. The hair was scanty, the nails brittle; the urine contained sugar in abundance. The findings were all more marked than in the younger brother. The children were both given thyroid gland extract with disappearance of the symptoms of myxedema and a complete clearing up of the glycosuria. When the thyroid treatment was temporarily stopped the apathy returned, and the sugar reappeared. The father of the children was neuropathic; the mother was obese and diabetic. However, the report of these cases finds nothing analogous in the literature; and no definite deductions should be drawn from this unique condition. It is well known that, as a rule, cretinism and other states of thyroid deficiency are associated with increased sugar tolerance rather than with glycosuria.

Infections.—Today, when almost all diseases are being associated with focal infections, it is natural to assume that some authorities claim diabetes to be in this class. The subject is too new to quote references or authorities and yet too interesting and important to neglect. Von Noorden[48] writing in 1912 sums up his own vast experiences by saying that there is no secure proof of a relationship between infections and diabetes.

In several of our cases in children, the disease unquestionably followed severe attacks of tonsillitis, but tonsillitis is so frequent an occurrence in children that one must be cautious about drawing conclusions. One of our patients, a boy four years old, gives a history dating very definitely from an attack of scarlet fever a few months before seeing us. This boy also had nephritis and was extremely sick. He did not remain under observation long enough for us to study the case in detail, and is the only patient in our series which has followed any of the acute exanthemata. Von Stärck[49] reports a case of a seven year old girl who developed diabetes mellitus in the course of whooping-cough. However, other workers have reported the onset of diabetes after exanthemata. Poor teeth and alveolar abscesses were very strikingly present in one young girl with a severe diabetes. After considerable improvement by the usual dietetic procedures, the disease reached a standstill. Thorough dental treatment resulted in a cleaning up of her mouth; and almost immediately following this, there was a marked and steady temporary improvement in her condition. One young boy who had moderately severe diabetes, also had enlarged and diseased tonsils. Frequent attacks of tonsillitis occurred, and each attack made the diabetes increase in severity. The possible causal connection was so apparent that we urged removal of the tonsils, but by the time the consent of the parents was obtained, the patient was already too ill to submit to the operation. In one of my older patients with mild diabetes and diseased tonsils, the removal of the tonsils resulted in a complete, although temporary, clearing up of the glycosuria.

To sum up the relation of the infections to diabetes, it seems to me that at the present time we are justified in seriously considering such a possibility in every case and in carefully searching for the presence of the focus of infection. When found, it is wise to remove such a focus, provided the condition of the patient does not contra-indicate operative interference. We do not feel convinced by any means that such procedure will relieve the disease, but it is surely true that a child with diabetes is better off if at the same time it does not have to combat another illness. This, perhaps, is of particular value when we realize the effect of diabetes in predisposing the child to various infectious conditions.

In children it seems to be especially true that the onset of an acute infection seriously increases the dangers of the diabetic condition. In one of my patients each attack of tonsillitis made the diabetes more difficult to treat.

Syphilis.—Although syphilis occasionally is an etiological factor in diabetes, the association of the two diseases is by no means common. I have never seen a case of diabetes in a syphilitic child, nor do the records of syphilitic children show that glycosuria is common. I have seen diabetes in adult patients who have had syphilis, and in these cases, the disease has been mild, but this is not a common experience. The probabilities are that, generally speaking, if syphilis is detected in a diabetic, the findings are coincidental rather than causal.

In 1916 Warthin and Wilson[50] published a report on the "Coincidence of Diabetes Mellitus and Syphilis" which, if verified by subsequent study, may compel a change of viewpoint. Performing autopsies on six diabetic patients they found that "the histological changes of chronic fibroid myocarditis and chronic aortitis of the type always associated with chronic syphilitic infection were present. The microscopic pictures presented by both these conditions are characteristic of syphilis, and would be sufficient in themselves to warrant a diagnosis of syphilis without the corroborating report of the other changes present and the demonstration of the spirochetes." In the pancreas they found a fatty atrophy associated with chronic interstitial pancreatitis and changes in the islands of Langerhans in all six cases: either a greatly diminished number of islands or a hyaline fibrosis. In one case spirochetes were found in the pancreas. Warthin concludes from his study that "latent syphilis is the chief factor in the production of the form of pancreatitis most frequently associated with diabetes, but that diabetes is not always coincident with severe degrees of this type of pancreatitis." Most students of diabetes do not concur in

Warthin and Wilson's view. Williams[51] has studied the question from the clinical and serological viewpoints and reports Wassermann tests on 143 patients, of whom only four showed positive reactions.

Dietary Excesses.—It would be extremely difficult to prove that diabetes ever followed dietary excesses. Yet it is striking that the disease is more common among classes of people who are good livers. One of our children gave a history of the onset of the diabetic symptoms shortly after a Christmas holiday in which she had over-indulged in candies and other sweets. But, of course, so many children over-indulge in dietary indiscretions during the holiday seasons and so few develop glycosuria that a single instance of this kind is of no importance.

Williamson[52] studied 300 cases of diabetes in adults and found great and prolonged excess of sweet foods and drinks in 31 per cent. of his cases. Knox (*loc. cit.*) calls attention to the fact that a few of the cases under one year of age very definitely followed over-indulgence in a poorly balanced diet too high in carbohydrate. For diabetes to develop in infancy from over-indulgence in food, it would be necessary to conceive of some congenital weakness of the carbohydrate metabolism, as has been suggested by Naunyn (*loc. cit.*). The cases of consanguineal diabetes reported by Foster[53] and some others reported by Morse[54] would add evidence to Weichselbaum's[55] idea of hereditary pancreatic weakness.

Obesity.—Obesity, which plays an undoubted rôle in the disease of adults, probably has no influence at all on diabetes in children. Williams[56] studied 100 cases of diabetes and compared their family history with the histories of 100 non-diabetic patients; and he found an overwhelming preponderance of relatives with obesity in the diabetic group. As far as children are concerned, obesity in the family history has not been noted as particularly common.

PATHOLOGY

Modern conceptions of the pathology of diabetes center around the pancreas, and in perhaps no other disease has experimental medicine contributed more to the advancement of the art of practice. Allen's[57] studies on the pancreas, performed with the precision of a mathematical formula, have followed along lines originally laid down by Minkowski and von Mering[58] in 1889. These earlier workers published articles that, read under the searchlight of the most recent investigations, still stand as authoritative as they were when first published. The original experiments dealt with complete extirpation of the pancreas with subsequent development of the most severe and fatal type of diabetes. Subsequent work by Minkowski[59] proved that partial extirpation resulted in mild or partial diabetes. Somewhat later, Sandmeyer[60] performed the operation of incomplete extirpation of the pancreas with ligation of the external pancreatic duct, and thereby produced a mild, delayed and slowly progressing disease, the so-called "Sandmeyer diabetes." In Minkowski's[61] second publication in 1893, he showed that the removal of seven-eighths or eleventwelfths of the gland resulted in a kind of alimentary glycosuria, comparable to the mild type of diabetes in man. One of his dogs developed glycosuria only after eating grape sugar, while a pig showed sugar only on a bread and meat diet, a reduction of the glycosuria on meat alone and complete freedom from sugar after three days starvation. Minkowski concluded from these experiments that partial removal of the pancreas produced a mild degree of the same disturbance which was observed to the highest degree if the pancreas were completely removed. He further suggested that the mild form of diabetes in man can be referred to a disturbance of the pancreas in the same way as the severe cases. Allen's[62] work, on which the form of treatment known as the Allen method is based, was done on cats and dogs. He showed that the removal of about nine-tenths of the pancreas resulted in the production of a severe diabetes, while the removal of seven-eighths of the gland produced a mild disease.

The pathologic picture presented by the pancreas left behind in the experimental animal is that of a peculiar type of degeneration in the Islands of Langerhans. This degeneration, it has been claimed, is identical with that found in the pancreas of humans who have died of diabetes, provided the autopsy is performed shortly after death and special methods of fixation and staining are employed. It is true that earlier statistics seem to show no constant pathological change in the pancreas of persons who have died of diabetes, but it is claimed that the pancreas is a highly digestive organ, and unless special precautions already mentioned are employed, histologic changes may be overlooked. It is also true that patients suffering with disease of the pancreas, for instance, chronic pancreatitis or carcinoma, frequently show no tendencies to either diabetes or glycosuria. This is probably due to the fact that the Islands of Langerhans, which have to do with the internal secretion of the pancreas, are not affected.

Although from a purely anatomic point of view the causal relationship between disease of the pancreas and diabetes is not absolutely established, further proof of this relationship may be obtained from a study of the course of the disease in animals. The earlier workers, as well as Allen, had been able to show a clinical similarity between canine and human diabetes, the importance of which from the standpoint of pathological physiology cannot be over-estimated. Some of Allen's (*loc. cit.*) dogs, in which he had produced a mild diabetes by partial extirpation of the pancreas, would remain in the condition of mild diabetes as long as their food intake remained under the limits of tolerance established for the animal. However, as soon as the limits were passed, the disease became progressively worse, and the pancreas at autopsy showed the same degree of pathologic change which was found in the animals originally made severely diabetic. On the whole, although we cannot absolutely say that human diabetes is caused by disease of the pancreas, the experimental and pathologic evidence so far brought together is practical if not absolute proof of this relationship. The recent studies of the Toronto group of workers[14] seems to add the final proof of the relationship of diabetes to the pancreas.

There are not many contributions to the pathologic anatomy of the pancreas in diabetes in children. Heiberg[63] performed autopsies on four diabetic children, ages, respectively, four and one-half years, five years, three months, and 15 months. Particular examination of the pancreas showed moderate atrophy of the whole pancreas and diminution in number of the Islands of Langerhans. Degeneration of the Islands was rare. In Knox's (*loc. cit.*) case the pancreas showed small round cell infiltration in the interstitial tissue, which in some places seemed to replace the acini. Connective tissue was increased; the Islands were diminished in size and number; and in one section no Islands were seen. The capsule was thickened, and trabeculæ were visible in the connective tissue. Martins'[64] series of 51 autopsies included four children, age 3, 12, 15 and 16 years. All of his cases showed histological alterations of the Islands of Langerhans, such as sclerosis, hyalin or hydropic degeneration. Not all of the Islands, however, are involved.

As for the exact method whereby carbohydrate control is lost in diabetes, there is little more than theory at present. Naunyn[65] was perhaps the first to suggest that diabetes is a functional disease, and Allen[66] has more recently emphasized the contention that originally, at least, diabetes is a functional disease of the nervous system, in that the average diabetic patient in the beginning has a normal pancreas. As the disease progresses, organic changes in the pancreas result from the constant overstrain in the same way that organic heart disease may ultimately result from what originally was functional overstrain.

It seems to us that in the light of modern knowledge it would be merely of historical interest to discuss any of the other theories which have been advanced concerning the pathogenesis of the disease.

METABOLISM IN DIABETES*

The metabolism of diabetes mellitus in children differs from the metabolism of adults only in the essential demands of the growing organism. A child of four years having attained a certain status in his diabetes will not long remain in this status, since his growing body constantly needs an increase in total calories as well as in protein. Probably the increased severity of the disease in children can be accounted for on this basis, since treatment at best can only hope to stabilize metabolic conditions at a fairly constant level. The inability greatly to improve the metabolic equilibrium offers a paradoxical situation for the diabetic child whose normal development requires a constantly changing metabolism.

One of the most apparent metabolic disorders in diabetes is the loss of weight despite a pronounced increase in food intake. Changes in the weight curve also occur under treatment. The loss of weight occurring as a symptom of the disease can be ascribed to a loss of body tissue. Although the patient may be ingesting enough food he is not burning enough to supply body needs, and his metabolic demands are met by inroads on his own body. There is rarely enough glycogen stored to affect weight, so that the fat supply is soon attacked and then the body protein. Marked changes in body weight may also occur as a result of changes in the water balance; a diet rich in carbohydrates causes an accumulation of water; one rich in fat may be followed by the excretion of water. The therapeutic use of alkalies may also be followed by increase in weight due to water retention.

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* This discussion of Metabolism was written by my colleague, Withrow Morse.

In considering the weight curve of the diabetic it is important to evaluate these possible causative elements. Although formerly it was a dictum of treatment not to allow the patient to lose weight, the advocates of the starvation method of treatment believe undernutrition to be essential for the maintenance of a metabolic level consistent with prolonged life.

Fundamentally diabetes represents the inability of the body to oxidize glucose. The subsequent course of the metabolic disturbance depends to a great extent on factors inherent to the nature of the disease and on extraneous factors such as diet. Minkowski (*loc. cit.*) and Allen (*loc. cit.*) showed that the dog made mildly diabetic by partial excision of the pancreas could be rendered severely diabetic by food intake in excess of his tolerance. In man there are also variable indeterminate factors which produce rapid metabolic disturbances in one patient, whereas in the next patient an entirely different metabolic course is followed. In an ordinary severe case of diabetes the disturbance of carbohydrate metabolism is followed by marked disturbance of protein, fat, and total metabolism.

The question of caloric needs of the diabetic patient has been the object of much study. Benedict and Joslin[67] concluded from a study of a series of severe diabetics that the metabolism in severe diabetics was 15 to 20 per cent. higher than normal; or in terms of calories per kilo body weight, the severe diabetic would require 37–38 calories as compared to the normal 30–32 calories. Naunyn (*loc. cit.*) had previously on clinical considerations assumed a lowered metabolism for diabetics, and other workers have failed to substantiate Benedict and Joslin's original conclusions. Allen and DuBois[68] pointed out that of 26 carefully investigated cases of diabetes the basal metabolic rates were normal in thirteen (13), increased in nine (9) and decreased in four (4). Most workers agreed that the determination of the metabolic rate is influenced by the condition of the patient and by his previous treatment, especially his previous protein intake. Wilder, Boothby and Beeler[69] report studies of the basal metabolism of 31 patients with diabetes. They find "few deviations of the basal metabolic rates of patients with uncomplicated diabetes from the DuBois normal standard that may not be attributable to factors which would be equally effective in modifying the metabolic rates of non-diabetic persons." The emaciated diabetic with a low caloric food supply like the emaciated non-diabetic will show basal metabolic rates 15-30 per cent. below normal. They also demonstrated that the ingestion of a high caloric diet or of an excess of protein will cause an increase in the metabolic level, whether the protein is derived from exogenous or endogenous sources, and that clinical improvement with increased glucose tolerance varied inversely with the basal metabolic level. The latter observation has been confirmed on two severe emaciated diabetics under our care.

Benedict and Joslin (*loc. cit.*) believed that the presence of acidosis tended to elevate the basal metabolism. Allen[70] reported a case which tended to prove this; but later evidence is not conclusive, Allen and DuBois (*loc. cit.*) Bernstein and Falta,[71] Wilder, Boothby and Beeler (*loc. cit.*).

Respiratory Quotient.—Lusk[72] has called attention to the difficulty of analyzing the respiratory quotient in severe cases of diabetes. The perverted metabolism of all food elements, the destruction of body fat and protein, the possible effect of acidosis make too literal interpretations of respiratory quotients not without danger. The first effect of diabetes on the respiratory quotient would result from the failure to oxidize glucose, the respiratory quotient of which is unity. Therefore since glucose is not burning the respiratory quotient of the diabetic would be depressed. The degree of depression as the disease becomes severe will depend on many factors. The patient in nitrogen equilibrium but losing weight from the destruction of body fat shows a respiratory quotient around 0.69 and 0.70; the more severe case burning his own body protein, but unable to oxidize the glucose fraction of the protein will show a still further depression. Lusk (p. 471) has calculated the protein respiratory quotient in complete diabetes with a D:N of 3.65 as 0.632.

Some practical therapeutic importance results from a study of the respiratory quotient of a diabetic under treatment. Wilder, Boothby and Beeler (*loc. cit.*) showed that the respiratory quotient rose as the patient improved clinically; Joslin[73] found that during the fasting treatment respiratory quotients are raised, and we have a small series of diabetics whose respiratory quotients were raised synchronous with clinical improvement from dietetic and specific (insulin) procedures.

Protein Metabolism.—Formerly it was the custom to replace carbohydrate in the diabetic diet by large amounts of protein, and in cases so treated a large excretion of nitrogen was found in the urine. At present when low protein diets are used the nitrogen excretion may be correspondingly low. Thus Newburgh and Marsh[74] were able to show nitrogen excretion as low as 2.4 gm. when protein intake was 36.8 gm.; or 1.2 gm. with protein intake of 28 gm. Von Mering and Minkowski (*loc. cit.*) found that in dogs rendered severely diabetic by removal of the pancreas the urinary nitrogen bore a definite relation to the amount of glucose excreted. The sugar excreted was derived from protein destruction. Lusk (*loc. cit.*) has performed numerous experiments to show that in severe diabetes sugar derived from protein may not be burned and that in complete diabetes when no sugar is burned the ratio of glucose excreted to nitrogen excreted is 3.56 to 1 (D:N ratio).

It has been calculated that 58 per cent. of the protein is available as glucose, whether the protein be derived from food or from endogenous sources. In calculating diets the glucose fraction of protein is of great importance; especially in the severer cases who are no longer burning ingested carbohydrate and who have become greatly emaciated through loss of body tissues. In these cases the katabolism of body protein will produce glycosuria.

The possibility that some of the end-products of protein metabolism in diabetes (leucin, tyrosin, phenylalanin) may yield acetone bodies has again been emphasized by Woodyatt[75] who tentatively estimates that 45.44 per cent. of the protein metabolized may be finally available as fatty acid. In the severe diabetic with acidosis the metabolism of protein undergoes a change from normal, namely, the deviation from the usual path of urea formation to an increased production of ammonia for the purpose of binding the acid products into less harmful, more easily excreted ammonium salts.

Acidosis.—When the normal individual is deprived of carbohydrate his urine will show increasing amounts of acetone, aceto-acetic acid, and β-oxybutyric acid. This is due to a disturbance in fat metabolism and is analogous to the condition of acidosis seen in diabetes. In diabetes carbohydrate is withdrawn from the metabolism, whether it is absent from the diet or is not oxidized. For some reason as yet unknown complete metabolism of fat occurs in an exact quantitative relation to burning carbohydrate; when the organism is not burning sufficient carbohydrate fat above a certain amount will not be completely oxidized. The result of this incomplete burning of fat is at first an increased excretion of fatty acid derivatives in the urine (acetonuria). As the products of incomplete fat metabolism (β-hydroxybutyric acid, aceto-acetic acid, acetone) accumulate in blood and tissues the chemistry of the body undergoes profound changes to combat the increasing acid accumulation. The blood and tissues in life are never acid, and to prevent acidification the circulating buffer salts and protein are called into service. The deviation of protein metabolism from urea to ammonia is also a defense mechanism against acidosis. The CO_2 of the blood is profoundly altered in acidosis, being lowered from a normal of 50-65 volumes per cent. to 30 or lower, depending on the degree of acidosis.[76,77] This is due to diminution in the quantity of bicarbonate of soda. The characteristic clinical picture of acidosis as seen in the air-hunger of Kussmaul and deepening insensibility passing into coma is caused by no other condition.

The precursors of the acetone bodies which produce acidosis are fat and to a lesser extent protein. Practically 90 per cent. of fat—exogenous or endogenous—is available as fatty acid; the protein derivatives, leucin, tyrosin and phenylalanin, may likewise be converted into fatty acid. Since acidosis is caused by faulty combustion of fat, it is necessary in the treatment of the disease so to regulate the diet as to keep a mathematical balance between the oxidizing glucose fraction (antiketogenic) and the precursors of acetone bodies (ketogenic fraction).

No better summary of the metabolism of diabetes can be made than by quoting from Lusk.[78] "This, then, is the worst picture of the perverted metabolism in diabetes. Sugar cannot burn, fat burns only as far as β-oxybutyric acid, and as for protein, a part of its amino-acids are converted into sugar and another part into β-oxybutyric acid, neither of which can be burned."

DIAGNOSIS

The diagnosis of diabetes mellitus in children can easily be established provided certain definite diagnostic standards are accepted. Not all cases presenting sugar in the urine are cases of diabetes, but all cases of diabetes at some stage or other show sugar in the urine. Not every substance that reduces copper solution is glucose; and the first rule in establishing a diagnosis of diabetes mellitus must be the proof that the reducing substance is actually glucose. In nursing infants a reduction may be caused by lactose, and occasionally an anomalous excretion of other reducing bodies is found. When a urine is found to react with the copper sulphate solution, the reducing substance must be further studied. Before calling it glucose it must be proved first that it is fermented by yeast into alcohol and carbon dioxide, second that it turns the plane of polarized light to the right, and third, it forms specific osazone crystals with phenyl hydrazin. None of these tests is difficult to perform, and all should precede the establishment of a definite diagnosis. Lactosuria occurs not infrequently in infants and young children, and probably many of the earlier cases of supposed cured diabetics were cases of lactosuria. Lactose also reduces copper solution and is dextro-rotary, but is not fermented by yeast and forms a different osazone crystal. Finkelstein[79] says that the presence of considerable ammonia in the urine interferes with the detection of lactose and galactose by the ordinary copper tests and advises boiling the urine before testing for these substances. Other reducing substances can easily be separated by means of the three tests just described.

Even the establishment of the reducing substance as glucose is not final evidence that the patient is a diabetic. Over-feeding of carbohydrates occasionally results in the so-called alimentary glycosuria, which is apt to be transitory or to disappear immediately with the cessation of the carbohydrate excess. Occasionally transitory glycosuria is encountered in young people which may be of unknown etiology and may come from certain drugs or from injury. Glycosuria not necessarily diabetic is also not infrequently found in diseases of the ductless glands.

Diabetes is a disease "in which the sugar burning capacity has been affected," and is not a mere overflow of glucose from excessive intake, nor is it characterized by the occasional presence of glycosuria; yet, at times, the earliest symptoms of diabetes are either alimentary glycosuria or transitory glycosuria, and every glycosuria in a child must be viewed with suspicion and carefully investigated.

Perhaps at the present time the most reliable single evidence of the underlying cause of glycosuria can be obtained from study of the sugar in the blood. Sufficient work has already been done to give the curve of normal under most physiological variations. The normal blood sugar value, while not constant for all individuals or even for the same individual at different times of the day, will be found to vary from 0.04 to 0.12 gm. per 100 c.c. The normal person will react to an ordinary carbohydrate containing meal by a rise in his blood sugar, but in the normal this rise will never exceed the high normal value of 0.12 (80).

The diabetic, especially if he is passing sugar and has not been under treatment, will certainly show after a meal a blood sugar curve definitely above the high normal value of 0.12. Even the diabetic, however, may show a normal blood sugar value if the specimen of blood is taken when the patient has been starved. Mosenthal, Clausen and Hiller[81] have recently shown that the "maximal percentage of blood sugar occurring in diabetic individuals on a carbohydrate free diet, or one containing a moderate amount of starch, can be obtained by making the determinations one to two hours after breakfast. They warn against erroneous interpretation of blood sugar determinations made in the afternoon or evening. Our own studies on the blood sugar in the diabetic from the diagnostic point of view substantially coincide with those of Mosenthal.

It may be advisable in certain early cases of glycosuria without definite hyperglycemia to study carbohydrate utilization by means of synchronous blood sugar and urine determinations, according to the method described by Hamman and Hirschman.[82] Our[83] observations made in the Nelson Morris Institute for Research would indicate that there is a distinct difference in the reaction curve between diabetes where "sugar burning capacity has been affected" and other glycosurias. A weighed amount of glucose, depending on the age and size of the child, is given on an empty stomach in the morning in water in the proportion of three to one, to which is added the juice of a lemon. The blood sugar and urine are examined immediately before the test is started and at half-hour intervals for two or three hours afterwards. Although occasionally a normal adult will react after 100 gm. of glucose with hyperglycemia and glycosuria, this reaction occurs within a half hour of the giving of the sugar and disappears immediately. The diabetic may show neither hyperglycemia nor glycosuria until after the first hour or even hour and a half, and the curve of reaction is likely to be ascending to a plateau with a slow drop. This latter curve is not absolutely pathognomonic of diabetes, but it certainly indicates a metabolic disturbance indistinguishable from diabetes.

From a clinical point of view it is sometimes necessary to employ the test of time in making the diagnosis. Developed cases have a definite clinical history: increased thirst, increased appetite, polyuria, loss of weight with glycosuria form a clinical syndrome which is unmistakable. Although the incidence of the various symptoms in diabetes will be discussed in detail shortly, it is well here to emphasize the fact that many diabetic children do not *complain* of diabetic symptoms and only admit the existence of such symptoms when direct questions are asked. Even adult diabetic patients do not consider increased thirst or appetite of sufficient seriousness to warrant medical attendance. In children many diagnoses of diabetes are missed because the parent considers the increased appetite merely as a developmental phase.

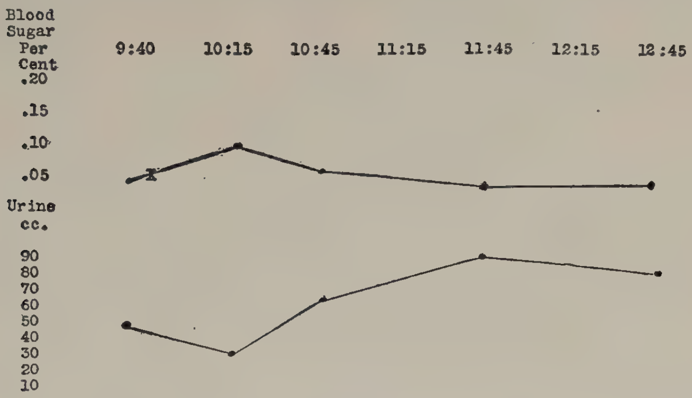

Fig. 141.—Chart 1. (Miss L.) A normal type of reaction. X, glucose, 100 gm. in 300 c.c. water.

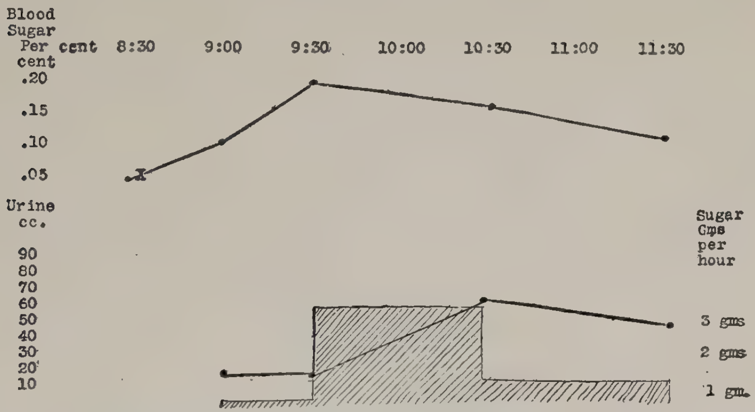

Fig. 142.—Chart 2. (G. H. L.) A diabetic type of reaction. X, glucose, 100 gm. in 300 c.c. water.

One type of glycosuria frequently mistaken for true diabetes is that formerly known as renal diabetes, but now more properly called renal glycosuria. In this condition, which is found especially in the young, sugar is constantly present in the urine, but without a definite relationship to carbohydrate intake and always associated with a low or normal blood sugar. It is very important to eliminate these cases of renal glycosuria because the condition is not a metabolic disorder and should not be treated as such. I have seen in the past two years at least four children presenting all the characteristics of renal glycosuria, in whose urine sugar is constantly present but who, despite the absence of any dietetic treatment have steadily shown normal development. In these cases, as well as in the case of a somewhat older person, the diagnosis of renal glycosuria was confirmed by the finding of normal blood sugar on repeated examination. Renal glycosuria is not admitted as a clinical entity by all clinicians. However, although I believe that all such cases must be viewed with suspicion until sufficient time has elapsed to develop into true diabetes, I am confident from my own experience that the disease exists and can be separated from cases of true diabetes mellitus. I have followed one such case ten years, and although the glycosuria has persisted none of the clinical symptoms of diabetes mellitus has developed. Two twins, age 11 years when first seen in January, 1918, were followed constantly for over a year, during which time sugar was constantly present in small amounts. The children were not put on any special diet. In fact, both were heavy eaters of carbohydrate food. The blood sugar of both children was below normal. In February, 1919, one of the children became sugar free; and in May, 1919, the other likewise became sugar free. Another case was that of a young boy seen first in 1916 at the age of 13 years, who showed sugar constantly for three years. In none of these last three cases did sugar reappear up to the time of writing, and at no time were any diabetic symptoms present. The last case was tested for his sugar utilization capacity three years after the onset, and a normal reaction was found.[84]

SYMPTOMS

The symptoms of diabetes noted in children depend almost entirely on how early the diagnosis is made. It is very probable that glycosuria appears before other symptoms are noted. Frequently, a marked loss of weight and lassitude are the most pronounced manifestations. As has been already stated, some children will show the increase of thirst, increased appetite and polyuria, which are so characteristic of the disease, and still go begging for a diagnosis because of the absence of urinalysis. The late complications of the disease, which are so common in adults are rarely, if ever, seen in children. Such things as carbuncles, which often lead the physician to suspect the presence of diabetes in an adult, are practically never present in children.

In Morse's[85] series of 19 cases in children, the symptoms were classified and divided as follows: Enuresis, 4, polydipsia 3, quietness 1, vision disturbances 1, emaciation 13, constipation 9, polyphagia 6, anorexia 3, irritation and itching of external genitalia 4, *polyuria in all.* Knox[86] calls attention to the changes in the appearance of the diaper as a frequent sign in infants. In older children fatigue on exertion, pruritus, eczema, teeth decay, headache, backache and radiating pains in the extremities are sometimes complained of. Abt (*loc. cit.*) lays stress on pains in the calf muscles as being particularly characteristic of diabetes.

COURSE

No description of the course of diabetes in children can be given without due consideration being paid to certain other elements in the disease. The course of the disease depends practically entirely on the treatment, and the treatment, as we shall discuss later, depends on the stage at which the diagnosis is made and the ability of physician and patient to coöperate with the object of obtaining perfect treatment. Hitherto, in practically all cases of diabetes in children, the course was steadily and progressively downhill, with the exception, perhaps, as Joslin (*loc. cit.*) has pointed out, of certain cases with a strong hereditary history. Treatment certainly can prolong life, even though the disease ultimately takes its course toward acidosis and coma. Almost all diabetic children have died of diabetic coma, often following over indulgence in carbohydrates. It is particularly to be marked that although diabetes in children may begin as a mild disease, the transition from mild to severe type usually occurs more rapidly than in adults. It is also true that lesser insults are required to disturb the metabolic equilibrium of the diabetic child than of the adult.

Only within the past few years has a somewhat more hopeful note been heard in the discussion of the course of diabetes in children. Joslin's[87] optimism for the future of the diabetic is nowhere more strikingly illustrated than in his remarks on diabetes in children. His statistics on 59 cases of children in the first decade of life show that 38 have died, that the average duration of life of all the fatal cases has been 1.4 years, and the duration of life in his living cases had already reached 2.6 years. To demonstrate the improvement in modern therapeutics he shows that of 15 living cases seen since Dec. 1, 1914, the average duration of life exceeded by nearly 50 per cent. the average duration of 30 fatal cases seen prior to that time. My statistics do not embrace nearly the number reported by Joslin, but they certainly show a steady increase in the length of life in the recent cases. One little girl, who gave evidence of being severely ill when first seen, lived for almost three years from the onset; a boy 10 years of age living under anything but ideal conditions lived at least two years and three months. Von Noorden[88] (1912) said that diabetes in children under 10 years seldom lasted longer than one and one-half to two years, and Joslin[89] quotes "a well-known diabetic author" as saying that "two years from the time of the first symptoms is the longest time that has come to my observation."

COMPLICATIONS

Complications of diabetes mellitus in adults may be grouped under two main headings; first those arising from trophic disturbances, associated with the continued state of poor nutrition of the tissues; second, acidosis and coma. Any other complications which arise are usually non-diabetic in origin, although they may, of course, exert an extremely harmful influence on the disease itself. The commoner trophic complications are infections, such as furunculosis and carbuncles, cataracts, and other disturbances of the special senses, arteriosclerosis and gangrene, caused by the arterial changes. This group of complications is rare in children.

On the other hand, the danger of acid intoxication and of diabetic coma continually threatens the child with diabetes. Perhaps the fact that the metabolism of the young makes greater demands and is less stable than the metabolism of adults explains the rapid course and the high incidence of diabetic coma before time has elapsed for the occurrence of trophic changes. Even in what is apparently a mild case of diabetes in a child a simple cold or an attack of tonsillitis may introduce the danger of acid poisoning.

From the side of the non-diabetic complications, tuberculosis always a threatening evil in the adult need not be seriously considered with the child. The onset, however, of any infectious disease always darkens the picture; for the lowered vitality of the diabetic makes him an easy prey to other diseases. For this reason, it is well always to consider the possible focus of infection in the diabetic child and whenever possible to remove such focus. As has been discussed under the heading of etiology, we cannot prove at the present time that focal infections play an etiological role in the production of diabetes, but we do know that the lighting up of an infection usually makes the burden of the diabetic that much harder to carry. Kleinschmidt[90] reports that in three of his cases measles had a decidedly unfavorable effect, and in one the disease was first discovered after measles. In a case reported by Naunyn (*loc. cit.*) varicella caused fatal acidosis; and in Störck's[92] case whooping-cough had the same effect.

TREATMENT

Graham Lusk[92] has said in discussing the scientific work which has been done in the study of diabetes mellitus that this work is "prophetic of possible accomplishment along scientific lines in the study of disease. It is typical of that scientific medicine which affrights the spirits devoted to a passing empiricism." In discussing the treatment of the disease which in the past has been so uniformly discouraging, we can well afford to discard the precepts of the passing empiricism and adopt the attitude of scientific medicine to which Lusk alludes. In other words, we need not accept as incontrovertible the statements made concerning the treatment of diabetes before the present era of intensive investigation started. May we not ask the question, Is it not possible that in the past we have overlooked or been unable to control certain more or less extraneous factors in the treatment of juvenile diabetes?

As far as we know today, diabetes in children is fundamentally the same disease as diabetes in the adult, modified only by the metabolic demands of the growing organism. This statement finds an almost exact analogy in other pathological states, for instance, heart disease. The cause and treatment of heart disease in the young differs in no essential detail from the cause and treatment of heart disease in adults. Even in heart disease, however, the difficulties of treatment in the young greatly exceed those found in adults, and this difficulty is probably due entirely or almost entirely to the physical demands of the growing organism. It is our purpose to discuss the treatment of diabetes in children from exactly the same point of view that we would discuss the treatment of the disease in older people; but we are compelled always to bear in mind the differences in their basal metabolism.

There is no single method of treating all cases of diabetes, and any hope of successful therapy must depend on a realization that certain basic principles must be established in each individual case. The first of these basic principles can be summed up in the word "individualization." By individualization of the patient, we mean a proper study for the purpose of classification. The child who gives a history of diabetes present for several months undiagnosed and untreated with a high glycosuria and acidosis offers a problem in therapeutics entirely different from the child whose disease is diagnosed early and in whom treatment can be begun at the very onset. Therefore, the first requisite for proper treatment must necessarily be proper study. The more important factors are detailed history, with special reference to family incidence of diabetes mellitus, previous history of the child, onset of disease and diet consumed since onset, a thorough physical examination, and then without change of the patient's habits an examination of a 24-hour specimen of urine. During these 24 hours, some responsible member of the child's family is urged to keep a detailed diary of the patient's activities.

The collection of the 24-hour specimen is important, and it is necessary to instruct someone how to do this. Perhaps the best and surest method is to save all urine voided *after* the morning meal and all subsequent specimens during the day and night, including the first specimen of the next morning. The whole is then measured. A simple and expedient method of measuring urine where more exact facilities are lacking is by the use of milk bottles, because it is comparatively easy for anyone to talk in terms of pints and quarts. Only by measuring the total excretion in 24 hours can the actual amounts of pathologic urinary constituents be estimated. For it is well to remember that the *percentage* excretion of sugar gives but a poor index to the *actual* metabolism. As an example, one patient passes five per cent. sugar, but his total urinary excretion is only 1000. The next patient passes only 2 per cent. sugar with a total urinary excretion of 3000. It is perfectly clear that the latter patient whose percentage excretion is only 2 per cent. is, however, excreting more sugar than the one who passes 5 per cent. In the more severe cases, where proper therapeutics is based on the exact knowledge of the source of the glycosuria, attention to the accurate measurement of the urine is of the most vital importance.

After the period of study and individualization, treatment may be begun along well-established principles but by means of a practice varying entirely with the nature of the particular case.

The first principle of treating diabetes is always the attainment of sugar-free urine. The means of accomplishing this object depend on the results of the preliminary analysis. Diabetes mellitus, although primarily, perhaps, a disease of carbohydrate metabolism which is manifested merely by glycosuria, ultimately becomes a disease of metabolism of protein, fat and carbohydrate with other manifestations besides glucose in the urine. The patient may show, then, merely glycosuria, or he may show glycosuria and acidosis, as has already been discussed under metabolism. The treatment of the extreme ends of this scale varies from simple reduction of carbohydrate in the mild case to the most careful control of all food intake in the severe case.

The preliminary analysis of a 24-hour specimen with the data gained from the study of the history will show approximately where the patient may be classified, and the diet for subsequent work follows almost automatically. The urine showing merely glycosuria and no acetone, possibly in a patient with no clinical manifestations of illness, centers attention mainly on gradual carbohydrate reduction. However, a urine showing in addition to glycosuria marked acetone reaction, ferric chloride reaction, increased ammonia, or other signs of acidosis as discussed in Chapter XXVIII, Vol. II, in a patient with a history of loss of weight, centers attention immediately on all the food constituents. In the latter case it is well-known that fat metabolism is fundamentally disturbed and that the acidosis results from errors in the fat metabolism.

Before giving a standard test diet, which is the logical procedure at this stage, it is well to emphasize certain general truths not always accepted as necessary facts in the treatment of this disease. First, in the treatment of a diabetic child as well as of an adult, one should always remember the great inherent danger in sudden withdrawal of carbohydrates from a patient accustomed to large amounts. Nothing is more conducive to the production of diabetic coma than suddenly to induct such a patient into a carbohydrate-free regime. Likewise, the substitution of large amounts of protein, especially in the form of meat, will work permanent damage to the diabetic organism even though there may be temporarily the appearance of improvement. In several of the children treated by us a really pitiable state of affairs existed when they were first admitted because of the high amount of meat protein previously allowed in their diet. Fats, in excess, as has been mentioned above, produce acidosis quickly in the diabetic child; and fats, too, will cause a glycosuria when the amount of carbohydrate in the diet alone will not produce glycosuria.

It is, therefore, apparent that after the preliminary analysis some further test diet, based on our findings and on the principles just outlined, will not be difficult to construct. The protein allowance of such a diet should be the amount necessary for a child of the size and age under treatment. The carbohydrate should never be reduced suddenly to the amount which might produce acidosis. In mild cases the fats can be sufficient to prevent loss of weight; in severe cases the fats require special attention before any other food element is changed.

For a detailed study of the energy requirements of the growing child, the tables of Benedict and Talbot[93] should be consulted.

For a child of 10 years, weighing approximately 60 lb. with no marked acidosis a diet can contain 70 gm. of protein, 100 gm. of fat and 50 gm. of carbohydrate yielding 1400 calories. This diet gives sufficient protein, sufficient calories, and a proportion of two to one fats to carbohydrate. A menu made upon a basis of these requirements is shown in Chart 1.

Chart 1

Breakfast:	
Grapefruit	½
Eggs	2
Bacon	20 gm.
Bread	25 gm.
Butter	10 gm.
Coffee	
Cream	15 c.c.
Dinner:	
Cream soup	90 c.c.
Roast beef	75 gm.
Spinach	100 gm.
Lettuce salad	50 gm.
Ripe olives	15 gm.
Butter	20 gm.
Wine gelatine	120 gm.
Supper:	
Lamb chop	50 gm. (1 chop)
Asparagus tips	50 gm.
Egg plant	50 gm.
Tomato salad	50 gm.
Bread	25 gm.
Butter	20 gm.
Diabetic custard	50 gm.

Protein 70, fat 100, carbohydrate 50. Calories 1400.

The patient is placed on this diet for two days and the next step will depend entirely on the reaction. A mild diabetic will not infrequently show a normal urine within 24 or 48 hours after the institution of this diet. If, however, sugar is present, simple and gradual reduction of the carbohydrate may result in a complete clearing of the urine. In such a case the first step in treatment has been made. If, however, the reduction of carbohydrate causes the occurrence of an increasing acetone reaction, the fats will have to be reduced parallel with the carbohydrates.

If on the other hand the preliminary investigations have shown the case to be of the severe type with marked signs of acidosis in the urine, immediate attention to fat intake is necessary. Even before the test diet is used, complete omission of all fat is ordered. After two days protein is omitted after which the carbohydrates are reduced until both urinary acidosis and glycosuria disappear. This brings the patient down to starvation level. The starvation of a diabetic as a phase of treatment of the severe cases will, beyond any other dietetic procedure, produce beneficial results when no other method has succeeded in clearing up the condition. By far the majority of diabetic children will have been rendered sugar-free and acetone-free by the procedures already discussed; but if either sugar or acetone bodies still persist, starvation is the method of choice. In children starvation is easily born, provided care is taken to put something in the patient's stomach at frequent intervals. During the starvation day plenty of water, three cups of weak tea or coffee, and 300 c.c. of clear meat broth containing salt are allowed. If alcohol is used, a simple schedule for a starvation day can be arranged, as follows:

Chart 2

7	Coffee	1 cup
8	Wine and seltzer, equal parts	180 c.c.
9	Broth	50 c.c.
10	Water	
11	Broth	
12	Wine and seltzer	
1	Broth	
2	Water	
3	Coffee	
4	Wine	
5	Broth	
6	Water	
7	Tea	
8	Wine	
9	Broth	

This starvation is persisted in until the urine is sugar-free, and as a rule, if the directions for preliminary treatment are followed, it will rarely require more than two days. If, however, the case is so severe that glycosuria persists after four days, the fast should be interrupted by giving 1 gm. of protein and 0.5 gm. carbohydrate per kilogram body weight for two or three days, and then repeat the fast for three days unless the patient becomes sugar-free earlier. It is, indeed, rare in children to have to continue intermittent fasting any length of time.

One means of interrupting the fast is by the insertion of the so-called egg-vegetable days, which makes the fast very much easier for the patient and at the same time does not materially change the prospects of treatment. The vegetables for such a day should belong to the group containing 5 per cent. carbohydrate, should be boiled twice and the water of the boiling thrown away. The menu for such a day is seen in Chart 3.

CHART 3

Egg-vegetable Day

Breakfast:	
Grapefruit	½
Egg	1
Coffee or tea	1 cup
Dinner:	
Clear meat broth	120 c.c.
Egg	1
Romaine salad	50 gm.
Tea or coffee	
Supper:	
Eggs	2
Spinach	100 gm.
Tomato salad	50 gm.
Coffee or tea	

Wine or whiskey according to judgment of physician.
Water as wanted.

In some severe cases, particularly those in which acidosis still persists or those in which the nutrition of the patient is such as to render continued starvation inadvisable, we can employ the principle established by von Noorden[94] in the use of oatmeal days. Von Noorden showed that in patients such as we have described, who have been refractory to treatment, oat gruel properly used is often successful in relieving both glycosuria and acetonuria. The potential and actual dangers of a high fat diet make the method of preparation of the gruel differ from von Noorden's original instructions, which called for a large amount of butter. Oatmeal gruel made from 1 oz. of the dried American oats (adult dose—estimate the child dose accordingly) cooked for a long time, will frequently cause the disappearance of the acetonuria and sometimes even of the glycosuria, when given three or four times in the course of a day with enough skim milk to make it palatable. In fact, as has been shown by Blum[95] and others, almost any form of carbohydrate is allowed in the diet. Joslin,[96] from his metabolic studies in the respiration chamber, does not believe that the oat gruel is oxidized, although clinical results would seem to justify its use in certain cases.

In all cases now, from the mild to the severe, we have accomplished the attainment of a urine free from sugar and acetone bodies, and the patient is now ready for the final state of treatment, namely, tolerance testing for all constituents in the diet and the establishment of a diet which will prevent the return of sugar or acetone. Generally speaking, in testing tolerance we reverse the procedure adopted in the first stage. In the mild case after a few days of sugar-free urine, carbohydrate is added just to the point of producing glycosuria, and the patient is allowed approximately 75 per cent. of this amount of carbohydrate. The amount of protein should never exceed the requirements of a child, and only enough fat is given to prevent loss of weight and not to produce acetonuria. Subsequently, more fat can be added and the demands of the growing organism met, provided the patient can handle fats.

In the severe type of case, as soon as the urine has remained sugar-free for two or three days, the patient is first tested for carbohydrate tolerance without adding anything else to his diet. At this stage it is necessary to consult some of the food tables found in simple books on diet. Vegetables of the 5 per cent. variety are first added, approximately at the rate of 5 to 10 gm. of carbohydrate a day until 50 gm. of carbohydrate are given, unless the sugar appears earlier, more if tolerance is higher.

After the fixation of the carbohydrate tolerance, protein is then added at the rate of 5 to 15 gm. a day in the form of lean meat, fish, chicken or egg-white until the patient is receiving enough protein for his age. No more protein is then added, but now fat addition is carefully begun, the rate depending on the previous acidosis. Usually 5 to 15 gm. a day, in the form of fresh washed butter, cream, bacon, the yolks of eggs, can be handled until the patient is receiving sufficient to prevent loss of weight without producing signs of acidosis.

This method of treating diabetics is open to the objection of being time-consuming and of over-emphasizing undernutrition. Newburgh and Marsh (*loc. cit.*) in 1920 therefore advocated the use of a low protein, low carbohydrate, high fat diet and reported cases to prove the value of their method. The most recent studies have had as their object the balancing of the diabetic diet. Contributions by Shaffer,[97] Woodyatt (*loc. cit.*), Wilder and Winter,[98] Strouse[99] have all attempted to evolve a diet for the individual patient based entirely on his own metabolic equation. Certain theoretical and practical considerations form the basis of these newer studies. Briefly stated these are: (1) the patient's caloric needs must be met. If not sufficient food is given, the patient will metabolize his own tissues and the end-results of his metabolism as seen especially in his acidosis, glycosuria and nitrogen excretion will represent total metabolism as contrasted to mere food metabolism.

(2) Nitrogen metabolism can be maintained if the diabetic diet contains 0.66 to 1.0 gm. protein per kilo body weight; provided sufficient carbohydrate and fat are burned to meet caloric requirements.

(3) The ratio of fat to glucose must be below the point at which a clinically significant acidosis appears.

The various workers have used different methods of calculating the food values. Shaffer and Wilder have studied the actual chemical metabolism of food mixtures. Woodyatt finds his food values from certain formulæ. The glucose value of the food equals 100 per cent. carbohydrate, plus 58 per cent. protein, plus 10 per cent. fat. The fatty acid value is 90 per cent. fat plus 46 per cent. protein and the fatty acid-glucose ratio is not to exceed 1.5–1. Strouse in figuring diets neglects the glycerol fraction of the fat and the fatty acid fraction of the protein, and estimates the glucose value of the food as 100 per cent. carbohydrate plus 58 per cent. of the protein.

Applying the results of these studies to the treatment of the diabetic child does away with many of the steps of the method discussed above. The patient is placed on a test diet as similar to his previous intake as can be estimated and allowed to stay on such a diet for 2 or 3 days or until metabolic equilibrium is reached. If such a diet were to contain protein 50 gm., fat 100, carbohydrate 50, calories 1300, and the 24-hour urine showed 35 gm. glucose, positive acetone, and 8 gm. nitrogen, the method of calculation of his metabolism would be as follows: (In order to simplify the procedure, only one set of formulæ will be employed. The results will vary with other formulæ, but the general principles of the procedure are the same).

The excretion of nitrogen shows nitrogenous equilibrium, so that endogenous sources of glucose from consumption of body protein need not be considered. The glucose *metabolised* equals 100 per cent. carbohydrate (C) plus 58 per cent. protein (P) of the food, minus the amount lost in the urine, or $50 + 0.58 \times 50 - 35 = 43$ gm. This figure 43 represents the glucose tolerance of the individual patient and forms the keystone of his food arch.

The total food to be allowed the child should be just enough at first to meet his energy requirements. The most valuable information on this subject can be obtained from the tables of Benedict and Talbot (*loc. cit,*). To the basal heat production must be added 10 per cent. for the specific dynamic action of food and an extra 10 per cent. or more for the body movements, depending entirely on the amount of exercise the child is having.

Knowing the weight of the child, his glucose tolerance and his total food requirements the diet is started by figuring his protein which can safely be put at 1 gm. per kilo. If the child weighs 30 kilos (66 pounds), protein (P) equals 30 gm. The glucose fraction (58 per cent. protein) equals 17.4 gm. To ascertain the amount of carbohydrate in his diet it is necessary to subtract the glucose fraction of the protein from the total glucose tolerance, or here $43 - 17 = 25$ gm.

The amount of fat which any diabetic individual can utilize is limited mathematically by the amount of glucose he is burning. If more fat is added to the diet than can be completely oxidized, products of incomplete fat metabolism are formed and produce acidosis. Using the glucose valuation as we have figured it, fat can safely be added so that it will total in grams from 2.5 – 4 times the grams of glucose being burned. In this case glucose equals 43 grams so that from 107 to 172 gm. fat could be used to meet the energy requirements. No absolutely fixed fat figure should be striven for; no more fat should be given than is necessary to meet caloric requirements. The actual amount will depend to great extent on the state of nutrition of the patient and the extent of acidosis. It must never be large enough to produce acidosis.

By this procedure the patient in a few days should be free from sugar and acidosis and should be in nitrogen equilibrium on a diet which for the time being requires no alterations. Later if tolerance increases as a result of a period of sugar-freedom the diet can be increased accordingly and the developmental needs of the child can be met.

Insulin.—The development by the Toronto group of workers of an active pancreatic extract for the treatment of diabetes already has changed much of our practice in the treatment of diabetes in children. Insulin is a highly potent therapeutic agent; it is injected hypodermically from one to three times a day, depending on the severity of the case and the individual reaction to the injection. It has the power of causing the diabetic organism to burn increased glucose in a direct mathematical ratio to the amount of insulin injected. This increased power of oxidizing glucose persists only while the insulin is being used: as far as known at present permanent increase in tolerance does not result.

In children the results are striking. It makes possible immediate desugarization on diets compatible with life. Children have been tided over threatening crises, and then have gotten along with very small doses or even with no insulin. The dangerous periodic upset of the diabetic child may be prevented by insulin. Many patients are steadily using insulin, are keeping sugar-free, and are gaining weight and strength like normal children. In severe acidosis and coma it is literally a specific, and it certainly is the only therapeutic agent which offers any hope for the diabetic infant.

The Use of Alcohol and Alkali.—At this point it is well to discuss the use of alcohol and alkali. Cammidge[100] in a recent review of the treatment of diabetic acidosis assumes that neither the question of alcohol nor alkali in the therapeutics of acidosis is settled. It has been the practice of most clinicians to give sodium bicarbonate in large doses by mouth, per rectum, by hypodermoclysis, or into the vein when acidosis seemed threatening to send the patient into coma. Erhman[101] showed that the alkali salts of beta-oxybutyric acid were more easily excreted in the urine than the unbound acid bodies. Joslin,[102] however, thinks that large doses of alkali may free from the tissues acetone bodies which do no harm, and he believes that patients are better off when not given bicarbonate of soda unless the previous treatment had included alkali therapy. My own personal experience would indicate that patients do get along as well without alkali as with it and that the mere removal of excessive fats will in most instances obviate the use of alkali. It cannot be denied that in severer cases of acidosis the use of bicarbonate of soda is apt to upset the patient's stomach. If, however, the physician believes in using alkali therapy, he certainly ought to employ it to the limit, that is, until the urine has become alkaline. As Sellards[103] has shown, the amount of bicarbonate of soda necessary to render the urine of an acidosis patient alkaline is exceedingly high. Where 3 to 5 gm. injected intravenously will make a normal urine alkaline, it requires 30 to 60 gm. for the patient with acidosis. Murlin and Craver[104] find a beneficial effect from the use of sodium carbonate given directly into the intestines through the duodenal tube in a 1 per cent. solution. However, they are of the opinion that the use of sodium bicarbonate may result in real harm rather than good.

Alcohol in the form of light wines, whisky or brandy has been extensively employed in the treatment of diabetes. At the present time there seems to be a disposition on the part of some authorities to do without alcohol. The author feels from his experiences, however, that alcohol has a distinct field of usefulness in the treatment of diabetes in certain stages. In the first place, it is an easily oxidized food of comparatively high caloric value, and frequently it apparently assists in the combustion of fats and in this way acts as an anti-ketogenic agent. The author has seen patients in the very severe stages of diabetes show definite clinical and urinary improvement when large doses of alcohol were added to their diet.

Coma.—There is, of course, no sharp line of demarcation between severe acidosis and diabetic coma. The treatment of the two conditions is almost identical except in the matter of degree. If the patient comes under observation with the signs of incipient or actual coma, then all efforts must be rendered towards averting death by coma. Slight drowsiness, change in the respiration towards air hunger, increased pulse, large amounts of acetone bodies in the urine, a high ammonia output* or if other tests point to an increasing acidosis, then the glycosuria can be forgotten. Even at this stage there is no unanimity of opinion about treatment. The stomach should be washed and the lower bowel emptied by enemata; the patient should be given large amounts of water and carbohydrate in some form. Von Noorden[105] has been an advocate of the use of milk in as large quantities as the patient can handle; others use oatmeal as described above. The writer has found that at this stage the use of levulose, by mouth, is in some instances successful in averting coma. Joslin[106] gives the following rules of procedure for the treatment of diabetic coma:

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* Methods for estimating acidosis and glycosuria will be found in Chapter XXVIII, Vol. II.

1. A special nurse is provided for the patient for both day and night.

2. The patient is kept in bed and warm, and every effort is made to allay nervousness and discomfort.

3. The bowels are moved by one or more enemata, but cathartics are avoided.

4. One thousand c.c. of liquids are given within each six hours (for adults). The liquids are given slowly, hot, as coffee, tea, thin broths, water. If so much fluid cannot be given by mouth, salt solution or tap water is to be given by rectum, and if this fails, the intravenous method is used. In order to secure an introduction of sufficient fluid in the first six hours, the cleansing enema at the beginning of the treatment should be followed after one-half hour by an enema of 500 c.c. salt solution in all cases as a matter of precaution.

5. If the patient has been accustomed to the fasting method of treatment, begin or continue the fast, but if he has been upon a full diet, give 1 gm. of carbohydrate per kilogram body-weight during the 24 hours in the form of orange juice or oatmeal gruel made with water; *whichever course is adopted, it is to be followed until danger is over.*

6. Sustain the circulation with the help of digitalis. Caffein may be given subcutaneously or as black coffee by the rectum.

7. Avoid alkalis. If such have been previously given, omit at the rate of 30 gm. a day.

The only addition which the writer would make to this treatment would be in the use of alcohol. Although Joslin[107] is of the impression that alcohol is not of great value, the author feels that alcohol does help, without definitely being able to prove his assertion.

When the signs of diabetic coma have passed, the patient is in about the same condition as the one who has been starved to the point of becoming acetone-free. If the method of combating the acidosis involved the use of carbohydrates, the patient will show glycosuria. For perhaps a few days, until all danger is past, as Joslin (*loc. cit.*) suggests, it is well to make no change, but after the signs of acidosis disappear the patient can then be starved until he is sugar-free, or a diet can be balanced to suit his needs.

Coöperation.—One feature in the treatment of diabetes often neglected in institutions is the necessity of studying the patient under conditions analogous to his daily life. Although certain stages of treatment require rest and warmth, as soon as possible the child should be allowed exercise, study and other normal pursuits, to the same extent as his usual habits require. When he leaves the institution there must be some member of his household who is able to coöperate with the physician and who must be trained to keep a detailed diary of diet, activities and urinary findings, and to make at least monthly reports to the physician. In children, if any success is to accrue from treatment, it is fundamentally necessary to secure the earnest and intelligent coöperation of either a nurse or some member of the family who can and will control the cooking, diet and activities of the child.

Without such coöperation there would be little use in attempting treatment of diabetes in children. It is not at all difficult with modern methods of treating diabetes in the hospital to teach a member of the family the fundamentals and the details which are required for subsequent intelligent carrying out of instructions. With an older child it has been possible occasionally to arouse enough interest to make him willing to play the game of getting well, and when he does so, no other can be a more capable assistant; but when the mother or some other member of the family must be the pivot around which treatment rotates, as usually happens, we must begin at the beginning and teach the fundamental principles, first, of food values and the meaning of protein, fat and carbohydrate to the diabetic; second, the use of food tables with special reference to the substitution of equivalent parts; third, urinary tests for sugar, for acetone, and for albumen; and fourth, diabetic cookery. It has been our practice to take the most available member of the family into our confidence as soon as the patient comes under observation and to explain in exact detail the conditions as we find them and as we hope to make them develop. This member of the family follows through the various states of getting the patient free from sugar and acetone, is taken to the laboratory to perform the tests, is taken to the diet kitchen and allowed to figure out the diet, and is taught the very simple recipes one uses for special diabetic articles of food. After learning all this, the member of the family is shown how to chart graphically the various findings, the patient's condition, and the changes in diet.

Blood Sugar in Treatment of Diabetes.—Although we have refrained from any discussion of the details of technique involved in following the diabetic, we wish to call attention to the importance of following the blood sugar curve as treatment progresses. Although not all changes produced in the blood sugar by treatment are fully understood, it seems safe to say that successful treatment connotes not only a sugar-free urine but a blood sugar approximating at least the upper level of normal. From personal observations it seems probable that food of any sort in the diabetic may raise the blood sugar, and we should attempt to remove the condition of hyperglycemia before discharging our patient from institutional care.

PROGNOSIS

In discussing prognosis of diabetes in children, scientific judgment must be tempered by philosophical understanding of the difficulties of accomplishing the very exacting conditions demanded by ideal treatment. A purely scientific prognosis might be given on a theoretical supposition that all conditions of treatment can be fully met. Under such conditions, for reasons already given in the body of this study, one can say with certainty that at present the diabetic child has a decidedly better chance than he had 10 years ago. With the improved method of treatment the average diabetic child today lives longer than he did in the past decade; and if it were possible to maintain ideal conditions for a long enough period of time in all diabetic children, it might be possible likewise to increase the span of life considerably more.

Unfortunately, however, the life of such a child cannot be lived within the walls of an institution or under the constant control of proper authorities. At times of delicate balance in the metabolism, the diabetic child may have to go to school or play in the street with other children. Any normal contact of this child with the world submits him to the dangers of nervous influences (excitement or worry) which might easily be the straw to break the back of his metabolism. Any normal contact, with a few pennies in his pocket, may bring him to the corner store and its alluring display of cheap candy or cake. The sugar hunger is likely to overwhelm him, and a carbohydrate spree results. Many of the children whom I have seen with diabetes have gone into coma after just such a carbohydrate spree.

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CHAPTER XXV

DIABETES INSIPIDUS

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It is to be conjectured that Thomas Willis, in 1682, recognized a polyuria which was not of the mellitus type, for he distinguished between saccharine and nonsaccharine diabetes. In 1794, Johann Peter Frank defined diabetes insipidus as a long-continued polyuria of a non-saccharine nature, which is not the result of a diseased condition of the kidneys.

Fitz, in his interesting contribution to this subject, cites two early recorded cases, one in Paris in 1789 and one in England in 1791. The first case is one of great curiosity. It was reported by de la Chassagne in the Paris "Journal" of May 1, 1789.

"Catherine Bonsergent has been remarked from her tenderest years; a burning thirst, a drought without example, with which she has been continually afflicted from the time of her birth, have always fixed on her the attention of personal observation. Her parents, after having entrusted the first care of her to a nurse, took her home when she was three years old. It was not long before they observed that an extraordinary quantity of water was consumed in the house, and at length discovered that their daughter drank, every day, to the amount of two pailfuls. In summer she drank the first water she could meet with, in winter she had recourse to snow and ice, and she was always careful to reserve as much as would enable her to drink abundantly during the night.

"At the age of 22 years, she married one, Gery, a cobbler, from whom she continued to conceal her complaint till after their marriage. She has had by him eight children, three of whom are still alive, and she is now pregnant with a ninth. What appears particularly extraordinary is, that during her lyings-in, instead of having recourse to such food and liquors as would seem most likely to strengthen her, she chooses rather, for the sake of satisfying her thirst, which at those times is more intense, to drink almost without interruption three or four quarts of coldest water. During the severe cold of last winter, this woman, who was then pregnant, drank to the amount of four pailfuls of water in 24 hours; and her husband being unable to afford the expense of such a consumption, was under the necessity of supplying her with melted snow and ice. This woman has never made use of any sort of strong liquors, and if she drinks only a single glass of wine, she feels an uneasy sensation in all her limbs and seems to be in danger of fainting. She is not dropsical; she even enjoys a pretty good state of health; she voids naturally all the water she drinks, but her urine is extraordinarily fetid."

The definition given by Frank will still hold. Diabetes insipidus is defined as a chronic affection, characterized by the passage of large quantities of pale urine of low specific gravity, free from sugar, albumin, and casts, and usually accompanied by an insatiable thirst (Futcher).

This affection is of rare occurrence. In 1888, Eichorst reported that of 35,942 patients admitted to the Zurich Hospital, only 17 (0.02 per cent. suffered from diabetes insipidus. Gerhardt found 55 cases (0.048 per cent.) among the 113,600 patients treated in the Charité, Berlin, during a period of 19 years. Futcher states that there were only eight cases among the first 106,000 patients admitted to the Johns Hopkins Hospital. Thus, of 255,542 patients there were 80 cases of diabetes insipidus, or about 0.03 per cent.

Diabetes insipidus, generally speaking, is a disease occurring in comparatively young persons. Thus, of 85 cases collected by Strauss, 57 were under 25 years of age, 21 under 10 years and 9 under 5 years. Stoermer (1892), who collected 133 cases, found that the disease is more apt to occur in the first 30 years of life. Van der Heyden, who collected 87 cases, found that 67 occurred before 40 years, 49 under 30 years, 26 under 20 years, and 7 under 10 years. A case has been reported by Dickinson in a newborn child.

Male adults are more likely to be affected than female, and boys more than girls (Stoermer, Külz, Futcher). Of 173 cases analyzed by Roberts and Van der Heyden, 126 were males and 47 females.

There is a tendency, it seems, for the disease to run in families, as has been observed by Gay, Gee, Lancereaux, and A. Weil. The latter has recorded a very extraordinary instance of the influence of heredity on diabetes insipidus. Of 91 members of four generations, 23 had persistent polyuria without any deterioration in health, namely, the great-grandfather, his three children, seven grandchildren, and 12 great-grandchildren. Of the 22 affected descendants of the original case, there were 11 males and 11 females (Futcher.) Lacombe described a family in which a mother, her three sons and a daughter suffered from the disease, while the mother's brother and his children were similarly affected.

It has been recorded by Trousseau and by Ralfe that it is not unusual to obtain a history of tuberculosis, syphilis, gout, glycosuria or albuminuria in one or both of the parents.

The pathogenesis of the disease is uncertain. The first question that presents itself, as Erich Meyer has pointed out, is whether the primary symptom of the disease is polydipsia (excessive thirst) or polyuria. Several attempts have been made to settle this point. In 1912, Reichardt asserted that in every case of diabetes insipidus there is a psycho-pathological moment which induces a primary polydipsia. That this is an important factor in many cases is not at all disputed by Meyer, as the latter author states: "It is possible to explain cases of polydipsia due to a diturbance of the pysche of the individual. . . . But when Reichardt points out that in a number of instances no psychical derangements can be observed and in spite of this, he denies that any other form of this disease is possible save the psychical, it is difficult to understand what he means."* Cases of primary polydipsia have also been reported by Schwenkenbecher and by Ellern in demented individuals.

It is possible that the nervous system plays an important rôle in the causation of this disease. Claude Bernard has demonstrated that injury of the fourth ventricle in certain animals is followed by a marked polyuria. This polyuria is a primary one; that is, it is induced in animals which have been thirsted and are poor in water (Finkelnburg, Jungmann and Meyer). The condition that results is a drying of the tissues, and even of the blood, if water is withheld from the animal. Jungmann and Meyer have pointed out that the phenomenon is caused by the stimulation of the floor of the fourth ventricle, and it abates as soon as the irritation ceases. Polyuria may also be induced by stimulation of the third ventricle and of the splanchnic sympathetic. Meyer concludes that this can be explained on the ground that a vasodilatation of the kidneys is induced, which causes not only an increased water elimination in the urine but also augmentation of the salt excretion.

These facts alone, however, cannot explain the phenomena of diabetes insipidus, and cannot be used as an argument against a possibility of an inherent functional weakening of the kidney to concentrate the urine (Meyer).

Another theory to explain the pathogenesis of diabetes insipidus has been advanced by Strauss and by Frank who considered the disease a manifestation of the disturbance of the internal secretion. In 1901, it was reported by Schäfer and his collaborators that on injection of an extract of the posterior lobe of the pituitary gland or an aqueous solution of its autocoids into a vein of an experimental animal, there was noticed a dilatation of the renal arterioles and a general expansion of the kidneys, as shown by the oncometer. Accompanying this expansion there was observed a remarkable increase in the amount of urine voided. As Schäfer states, it might at first be supposed that the increase of urine is simply determined by the great increase of blood flow through the organ consequent upon the general vasoconstriction and the local vasodilatation which pituitary extract induces, but it may be seen that increased rate of flow is still maintained after the blood pressure has come back to the level at which it stood before the injection. Moreover, the effect of a second and subsequent doses of the autocoid, administered soon after the main result of the first dose has passed off, again produces an increase in the urine flow, although the blood pressure as a result of these after-doses does not rise, or even falls instead of rising, and although the kidney volume may now be unaffected. This experiment clearly demonstrates that the autocoid may affect not only the blood-vessels of the kidney but also its secreting cells, which it seems to render more active or more permeable. The urine secretion must, therefore, in these circumstances be induced by a direct chemical excitation of the renal cells by the autocoid. Schäfer emphasizes the fact that the diuretic action of the pituitary hormone is not antagonized by atropin, which may be taken as a sign that it does not act through the vagus nerve endings but directly upon the kidney cells.

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* Free translation.

Cushing in 1913 has demonstrated that section of the posterior pituitary lobe, or transplantation of the posterior lobe, induces a polyuria, and he assumes that human diabetes insipidus can be ascribed to some hypophyseal involvement.

On the basis of his own findings and those of Koranyi, Tallquist and Socin, Erich Meyer concluded that the disturbance in diabetes insipidus was due to an inherent functional inability of the kidneys to excrete urine of normal concentration; whereas Forsbach and Weber considered diabetes insipidus as the result of stimulation of the kidney by the intake of abnormal quantities of substances which are excreted in the urine.

Clinicians divide cases of diabetes insipidus in two groups: (1) The primary or idiopathic class, which includes the hereditary cases, and all those cases where there is no demonstrable organic lesion or basis for the disease; and (2) the secondary or symptomatic class, including the cases in which there is evidence of organic disease either in the brain, pituitary, sympathetic system, or elsewhere. Futcher, for example, is impressed with the importance of cerebral syphilis as a cause of this affection, and such cases are reported by Fournier, Oppenheim, Spanbock and Steinhaus, and others. Tumors of the brain may induce this affection. Cushing reports a case with a urinary output of 10 to 12 liters in a man 22 years of age, in whom autopsy revealed a lympho-sarcoma of the cervical glands and an infiltrating growth occupying the pituitary infundibulum. Hadra and Ralfe have reported cases accompanying aneurysm of the carotid and abdominal aorta respectively.

Tumors of the abdomen may be accompanied by diabetes insipidus. Dickinson found this affection in a patient with carcinoma of the liver. Schapiro reported five cases with chronic disease of the intestines who had a polyuria. Affections of the cord may induce this disease; thus Schlessinger found it in a patient with lues, and Schultze in a patient with a spinal cord tumor.

Diabetes insipidus may follow the infectious fevers, for example: Typhoid, typhus, diphtheria, measles, and scarlet fever (Futcher). Rarely, however, diabetes mellitus may pass over into diabetes insipidus (Senator).

Symptoms of the disease are those of the polyuria and the polydipsia that are so characteristic of the affection. The polyuria may be very marked, sometimes extraordinary quantities being voided in the 24 hours. It is the literal truth to say in some of the cases that the patient urinates pailfuls of water daily. Trousseau reports a case 24 years old who eliminated 43 liters, about 11 gal., a day. A remarkable instance is reported by Vierodt of a child weighing 13.2 kg., who voided 12.3 kg. daily. Futcher had a case who voided 16.5 liters daily, micturating 1720 c.c. at a single voiding. The composition of the urine will be discussed later.

The seemingly unquenchable thirst is a very pronounced symptom. Sometimes it is the first thing that calls the attention of the physician to the disease. One of Futcher's patients expressed it, that "he simply lived to drink," and described the infinite satisfaction it gave him to be able to take a whole pitcher of water at one draught. Trousseau's patient drank 50 liters (about 13 gal.) in a day. In demented individuals, or in those whose fluid intake has been restricted, the patient may drink his own urine (Strubell). Cases which show a primary polydipsia obey, according to Buttersack, the following requirements: (1) There is a normal sweat secretion. (2) The urine is less than the amount of fluids injected. (3) The polyuria ceases on restricting the fluid intake. (4) The urine elimination and liquids taken run parallel (Futcher).

The appetite, however, does not go parallel with the thirst. Sometimes it is diminished, while at other times, as in Trousseau's patient, it may be ravenous. The perspiration is decreased. Constipation is common, but there is no intestinal putrefaction. In the symptomatic cases, there may be a marked degree of emaciation, but not so in idiopathic patients.

The secretion of saliva may be diminished. The mouth is dry, and this condition undoubtedly adds to the insatiable thirst (Coleman). Külz, however, has observed increased secretions of saliva. Still has observed enuresis in children suffering from this disease, which he thinks is due rather to the rapid filling of the bladder than to any irritating quality of the urine. Holt calls attention to the nervous manifestations that may be present, such as headache, palpitation, flushing of the face, and other vasomotor disturbances and neuralgias.

The metabolism in diabetes insipidus has been studied by a number of authors. The state of nutrition in this disease does not, usually, undergo any important change. Magnus-Levy found that a patient weighing 65.5 kg. had an oxygen consumption of 210 c.c. a minute, which is equal to 3.97 c.c. per kg.—a figure somewhat above normal. Tallquist reports a case where the patient's weight was 65 kg., the gross intake 43 calories per kg., in which the increase of weight in four weeks amounted to 2 kg. During another period of examination, when the food contained 39.1 calories per kg., the weight sank from 66.8 to 62.2 kg. This decrease in weight was traced to loss of water, because, (1) it took place simultaneously with retention of nitrogen, and (2) the diuresis increased owing to the higher percentage of protein in the food. Tallquist found that with 43 calories per kg. of body-weight, and upon a diet poor in protein, there was an increase of 3 kg. in one month (Mohr). Similar results were reported by Vannini, Hirschfeld, and Butler and French.

The metabolism of protein has been studied by Strubell, Tallquist, Vannini, Gerhardt, Seiler, Butler and French, Rosenbloom and Price. In general it may be stated that the protein metabolism undergoes no deviation from the normal. Rosenbloom and Price reported that the partition of nitrogen in the urine was entirely normal. They found that the urea nitrogen was about 85 per cent. of the total nitrogen, the ammonia nitrogen was about 5 per cent., the amino-acid nitrogen 1.5 per cent., the creatinin nitrogen 2 per cent., the uric acid nitrogen about 0.2 per cent., the purin nitrogen 0.2 per cent., and the undetermined nitrogen about 6.1 per cent., of the total nitrogen.

Rosenbloom and Price observed a very low nitrogen loss in the feces, which shows a very complete absorption of the nitrogen intake, due, they think, to the increased ingestion of water, which Hawk and his corroborators have shown lessens the amount of fecal protein excreted.

Fitz, in his study of a case of diabetes insipidus, concluded that the renal function tests indicated that the kidneys as a whole were normal, but showed that there was present a vascular hyposthenuria with vessels hypersensitive to chloride stimulation. It has been found by Erich Meyer and others that the kidneys in diabetes insipidus have lost their power to concentrate the urine. When a normal individual on a standard diet containing a definite quantity of sodium chlorid receives in addition from 10 to 15 gm. of sodium chlorid, the salt concentration in the urine is increased, while the total volume of urine eliminated varies very slightly. In a patient with diabetes insipidus, however, an increase of the sodium chlorid of the diet does not increase the sodium chlorid concentration in the urine, but on the other hand increases the volume of urine voided (Rosenbloom and Price). It will be seen from the table taken from the work of Erich Meyer and of Rosenbloom and Price that the kidneys of normal and diabetes insipidus patients act differently in this regard.

In diabetes insipidus, the discharge of water through the kidneys is above normal, both absolutely and relatively. Normally, whatever the amount of fluid ingested, it is so distributed among the excretory tissues that between 66 and 70 per cent. of the water is discharged through the kidneys, the rest being carried off through the lungs and skin (*perspiratio insensibilis*). In the diabetes insipidus patient the kidneys remove as high as 90 per cent. of the total water excreted. The skin evaporation is, of course, lessened, so that one of the troublesome symptoms noticed by a patient may be his inability to perspire (Mohr). Strauss has found a higher osmotic concentration and a greater salt content in the prespiration than in the urine ($\Delta = -0.40$; NaCl = 0.41 per cent.). This is exactly the reverse of the normal state, where the freezing point of the urine is invariably higher than that of the perspiration (Mohr).

TABLE 1.—CHLORIDE METABOLISM IN NORMAL AND DIABETES INSIPIDUS PATIENTS

Volume urine, cubic centimeters	Specific gravity	Δ* of urine	NaCl in urine		NaCl intake, grams	Author
			Per cent.	Grams		
1375	1.011	0.78	0.198	2.72	. . .	Meyer†
1200	1.011	0.91	0.444	5.38	1.0	Meyer†
1700	1.012	0.88	0.374	6.36	. . .	Meyer†
1450	1.010	0.75	0.316	4.48	. . .	Meyer†
1890	1.005	0.24	0.110	2.08	3.4	Rosenbloom and Price‡
3080	1.005	0.22	0.110	3.38	3.5	Rosenbloom and Price‡
3080	1.005	0.25	0.108	3.32	9.5	Rosenbloom and Price‡
4000	1.003	0.27	0.102	4.10	3.4	Rosenbloom and Price‡
5190	1.002	0.22	0.103	5.36	3.4	Rosenbloom and Price‡
4305	1.002	0.26	0.109	4.70	3.4	Rosenbloom and Price‡
4400	1.002	0.28	0.100	4.40	3.4	Rosenbloom and Price‡
4260	1.003	0.24	0.109	4.66	9.6	Rosenbloom and Price‡
5500	1.002	0.22	0.101	5.56	3.5	Rosenbloom and Price‡

*Freezing point of urine.

†Normal case.

‡ Case of diabetes insipidus.

The urine, on the whole, is watery, has a low specific gravity approaching that of water and may contain inosite. This has no special significance, since it has been found in other diseases than in urines of healthy persons who have partaken of excessive quantities of water. There may rarely be a trace of glucose or albumin, the former due to an alimentary glycosuria, and the latter to a chronic irritation of the kidneys.

The sulphur partition, as studied by Vannini and by Rosenbloom and Price, shows no increase in the ethereal sulphate fraction (thus indicating the absence of an intestinal putrefaction) and no change in the neutral sulphur portion, indicative of no reduction of tissue oxidation. The metabolism of phosphorus, calcium and magnesium were found normal (Rosenbloom and Price).

The blood shows very few changes. The specific gravity of the blood was found by Stoermer to be increased —1066— but Strubell, Gerhardt and Strauss found normal figures from 1055 to 1060. Meyer has found a greater number of erythrocytes and a higher percentage of hemoglobin. I have had occasion to examine the blood for the following constituents in a case of diabetes insipidus with these results: Carbon dioxide combining power of the plasma, 56 per cent.; urea nitrogen, 19 milligrams per 100 c.c.; non-protein nitrogen, 28.5 mg.; creatinin, 0.85 mg.; uric acid, 2.45 mg.; cholesterol, 150.5 mg.; and glucose, 0.085 per cent. These figures are normal.

The course of the disease is usually of long duration. Cases are on record, however, which run a very rapid course. In the symptomatic type of cases, the duration of the disease depends on the cause. Thus, for example: If the disease is due to syphilis, a rapid cure may be obtained by treatment, whereas if it is due to some tumor in the brain, the course of the disease is the course of the etiological factor. In the idiopathic cases, the disease has been known to persist for 50 years (Coleman), and Weil has reported cases lasting 83 years, 87 years, and 92 years respectively (Umber).

The prognosis of the disease in most cases is favorable as to the continuance of life; it is unfavorable as to the cure of the disease. The outlook depends upon the causative factor. Thus, Strumpell states that if syphilis is the cause, the disease will yield to specific treatment. Meyer writes that the loss of water *per se* is a source of no danger to the body, neither to the circulatory system nor to the general condition of the tissues. If the disease is caused by some severe disturbance of the central nervous system, the prognosis is, of course, unfavorable.

The treatment of diabetes insipidus has for its object two purposes: (1) The treatment of the causal disease, (2) the treatment of the symptoms. The first course of procedure depends upon the underlying disease. If the patient is suffering from a psychosis, psycho-therapeutic measures should be resorted to, such as hypnosis, suggestion, psycho-analysis, etc. In definite or suspected cases of lues, anti-syphilitic treatment with salvarsan, mercury, and potassium iodid should be administered. In many cases, it is difficult to say whether the anti-luetic treatment has acted because of their anti-spirochetal properties, or whether the injections serve as a suggestive treatment to the mind of the patient. Erich Meyer thus criticizes the report of the results obtained by Benario with salvarsan treatment.

Recently the endocrine treatment of diabetes insipidus has taken a great step forward, and though the enthusiastic expectations of certain authors have not been realized, still progress has been made. In 1913 von der Velden found that in human beings with normal kidneys, the injection of extracts of the posterior lobe of the pituitary gland did not increase, but, on the other hand, lessened the volume of urine excreted. In a case if diabetes insipidus he was able to cause a marked concentration of the urine by the administration of posterior pituitary extract. Bab, in 1916, and Motzfeldt, in 1917, similar results were obtained.

Barker and Mosenthal found that the most favorable effect was obtained when two or more injections of not less than 1 c.c. each of the preparation of posterior pituitary extract were given during the course of the day. They found that the volume of urine was reduced, its specific gravity raised and the percentage of sodium chlorid and of nitrogen in the urine increased. According to these authors administration of the gland by mouth was without any effect. It seems that tethelin and adrenalin have no value in the treatment of diabetes insipidus. Their results were confirmed by Rosenbloom, who found that the thyroid gland does not influence the polyuria.

The dietetic treatment is very important. Erich Meyer advises the choice of a diet which should least irritate the kidneys. The substances that stimulate the kidneys and which, therefore, should be avoided are sodium chlorid and urea. The reduction of both of these substances in the diet lessens the thirst. It is known from the work of Stahelin and Anderson that a vegetable diet lessens the volume of urine. A diet composed mainly of carbohydrates and fats with a minimum of protein can be prepared. Such a diet is poor in salt and still not unappetizing. If to this diet are added fruit, etc., which are rich in water, it is possible still further to cut down the pure water intake. It is essential that the diet be carefully prepared. The salt content should be painstakingly determined as in a nephritic diet, but here it is permissible to add a few condiments, like vinegar and mustard. After the patient has been kept on such a diet for a long time and his urine excretion has been markedly diminished, it is possible gradually to add other constituents to the food. The periods of feeding should be carefully arranged. It is necessary that the supper should not be late and should contain the least possible quantities of salt, etc., so that the patient will not be disturbed during the night to void urine or to quench his thirst.

The diet may consist of green vegetables, fruits, puddings, pastry, unsalted bread, unsalted butter, and meat without salt. The sodium chlorid content should be kept as low as possible. The following table, taken from Riesman, giving the sodium chlorid content of the principal foods may be useful in preparing diet lists.

Sodium Chloride Content of Foods (Lava)

Food, 100 gm.	NaCl, gm.
Various kinds of meat	0.09 – 0.17
Fresh water fish	0.06 – 0.12
Salt water fish	0.16 – 0.41
Poultry of various kinds	0.14 – 0.17
Oysters	0.52 – 0.14
Caviar	3.00 – 6.18
Smoked and salted meats and fish	1.85 –20.59
Bacon	1.0
Ham	1.85 – 7.50
Fish in oil	1.79 – 5.49
Sausage	2.20 – 8.10
Canned soups	8.10 –15.48
Meat extracts	1.40 –14.60
Commercial sauces	9.37 –22.46
Eggs	0.13 – 0.21
Egg—white	0.31
Egg—yolk	0.39
Milk	0.15 – 0.16
Cream	0.012
Unsalted butter	0.02 – 0.21

FOOD, 100 GM.	NACL, GM.
Salted butter	1.0 – 3.0
Oleomargarine	2.19
Cheese of various kinds	1.59 –10.57
Bread	0.5 – 0.6
Cereals	0.014– 0.046
Flour—various types	0.002– 0.35
Oatmeal	0.26 – 0.29
Potatoes	0.016– 0.078
Legumes	0.058– 0.0
Salad	0.17
Cauliflower	0.15
Spinach	0.21
Celery	0.49
Canned vegetables	0.67 – 1.27
Sauerkraut—pickles	0.73 – 1.45
Fruits—various kinds	0.004– 0.07
Almonds	0.09
Walnuts	0.19
Mustard (table)	2.66
Tea, coffee	0.05 – 0.15
Ale	0.10
Ordinary drinking water	0.002– 0.23
Soups	0.3 – 1.0

It is advisable to administer certain sedatives to give the patients good nights' rest. Meyer advises the use of veronal, others recommend the opiates. These narcotics, as has been pointed out by Frey and Kumpiess, lessen the urine elimination but do not influence the concentration of the urine.

Certain authors have recommended the use of strychnin nitrate (0.005 to 0.01 gm. *pro die*). Umber as well as Meyer has never observed good effects with this alkaloid.

The sense of thirst may be lessened by the opiates. The crude opium or extract, in half-grain doses three times daily, gradually increasing until a total of 4 to 6 gr. daily are taken, may be tried. Codein may be administered in the same way. Valerian is much used. Five grains of the powdered root, three times daily and increased to a total of 2 dr., may be tried; or the zinc valerianate in 15 gr. doses, increased to 30 gr. three times daily, may be the choice. Some authors have used ergot—10 minims of the tincture of fluid extract three times daily. Futcher states that this is not infrequently followed by a reduction in the volume of urine. Erich Meyer has found that theocin increases the functional activity of the kidneys. It should be given in 5 gr. doses three times daily.

Herrick reports a case in which he observes a remarkable reduction in the urine excretion after removal of 5 c.c. of cerebrospinal fluid by lumbar puncture. In a case of traumatic origin, Bach applied ultra violet rays to a neck scar, the result of an abscess that had developed following a mastoid operation. The thirst and the polyuria lessened and finally disappeared.

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CHAPTER XXVI

SEASICKNESS

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Seasickness, a disease which often occurs among people voyaging at sea, is characterized by a definite syndrome of symptoms, of which nausea, vomiting and mental depression are the most marked.

The first detailed report of seasickness is found in the writings of Hippocrates who said in part, "sailing on the sea proves that motion disorders the body." The disease was mentioned by Homer, and reference is made to it in Horace and Virgil. The relationship of visual disturbances and seasickness was discussed in 1757 by Gilchrist; and later Darwin in 1794, Miller in 1808 and Bell in 1826 wrote of this association. Allard in his *Thése pour le Doctorat, à Montpellier*, 1829, claimed that a very definite association existed between visual impressions and seasickness. Early in the nineteenth century Wollaston's theory of brain hyperemia attained considerable prominence, and some years later Aronsohn suggested practicing on an oscillating plank before going to sea as a prophylactic measure against the disease. That there are so few references to the disease among the older writers may be accounted for by the fact that traveling by sea in very early times was dangerous, and people who were compelled to travel in this manner were so expectant of the grave perils that they had little thought for the physical discomfort which sometimes attended their journey.

Since we are without definite knowledge of the specific cause of seasickness it is only natural that there should be many different theories concerning this illness. Some theories are based entirely upon psychic disturbances, in which the elements of fear, imagination, and the influence of the will receive prominent consideration; while other theories are founded on purely somatic factors, physical or chemical; and combinations of the psychic and somatic theories are numerous.

Adherents of the psychic theories maintain that fear predisposes to seasickness, because in fear a depression of the higher brain centers with consequent derangement of the cerebral function is manifest. In support of this it has been shown that a sufficient effort of will can prevent the occurrence of the disease. Nagel asserted that appreciable external or internal stimuli are not essential to produce the symptom complex of seasickness, but that imagination may set up a molecular shifting of mass in different organs which brings on the symptoms of the disease. Other observers in support of the psychic theory draw with the stroke of finality a contrast between the happy, contented baby and the mentally depressed, apathetic, ill mother. The baby has no fear because the dangers of the sea are unknown to him, and the rolling of the ship gives him rather a sensation of pleasure. This fails to explain, however, the not uncommon occurrence of seasickness in children of five or six years who are unaware of the dangers of sea voyage and who are fond of swinging; nor does it take into account the fact that many older children anticipate with delight the first journey on the sea.

The various theories based on somatic factors consider chiefly the physical factors of external stimuli and mechanical influences. Of these influences the motions of the ship are of most importance. A ship's motion occurs around two axes, the transverse and longitudinal. Motion occurring about the transverse axis is greatest at the bow and stern and least amidship. It is the most marked motion of the ship and is analogous to the action of a "teeter totter." A rolling motion occurs around the longitudinal axis, and is appreciably less than that around the transverse. Other motions are: lifting and falling, and a combination of roll and pitch.

According to Rosenbach if a body receives a strong impulse too suddenly, without a precursory wave of irritation which reflexly indicates to the adaptive organ the amount of work to be expected, then all measures having the function of meeting this signal wave are incomplete because sufficient preparation is impossible. This phenomenon is particularly applicable to the individual aboard ship. When the body suddenly and without previous indication of the amount of work to be done by equilibrium receives acceleration in an upward direction and just as unexpectedly sinks downward, the forces which are able to lessen the shock of the unexpected motion cannot function properly. Even though a signal reflex wave is received to equilibrate the body to the first motion, the response may be so delayed that it will be interfered with by the opposite rapidly following external stimulus to which the body is involuntarily subjected. On account of these unusual processes the impulses by which the different organs are notified are not carried to them normally.

Seasickness may be said to belong to a group of affections dependent in part for their symptoms upon sudden kinetic disturbances. It is the foremost representative of this group and is comparable with less intensive static disturbances, such as merry-go-round riding, riding in elevators or street cars, rapid turning in a circle, rapid dancing, standing on one's head, or riding backwards.

The theory of brain hyperemia was first advanced by Wollaston as a cause of seasickness. He compared the blood-vessels to a mercury barometer without considering that the vessel walls are elastic and are kept in a state of tonus by the vasomotor system. Certainly more plausible are the theories of brain anemia or of changes of anemia and hyperemia. When one considers the symptoms of apathy, pallor and feeble pulse and the fact that these symptoms are greatly lessened when the patient lies down one is inclined to favor the theory of brain anemia.

An explanation of the pathogenesis of seasickness was attempted by Darwin, by Bourru and many others on the hypothesis that the disease was caused by visual vertigo. These observers assumed that in the maintenance of equilibrium, visual impressions and the muscle joint and tendon sense are involved with the center of equilibrium in the cerebellum, and that a disturbance of this mechanism occurs in seasickness. The motions of the ship prevent one from focusing one's eyes on a fixed point and seeing other objects in relation to it, resulting in a disorientation which is increased by cerebral anemia and vertigo. Castelli claims that such visual vertigo is the underlying factor in seasickness, and asserts that it is the same phenomenon observed in a person on top of a tower whose eye adjusts itself to all the different horizons that can be seen from the top. The eye of the observer changes every few seconds to a different horizon and the person has, in consequence, the sensation of loss of equilibrium.

Such visual vertigo theories are entirely untenable since they do not explain all phases of the disease. While there is no doubt that external stimuli may play a part in the pathogenesis in some cases, it is equally certain that external stimuli such as visual impressions are not necessary to produce seasickness. Visual vertigo theories do not take into account the fact that blind people become seasick; and that the disease continues even though the eyes are kept closed, and when the patient remains in darkened quarters.

Larrey assumed the condition to be one of cerebral concussion; while Foussagrives thought of it as a disturbance due to vibrations of the contents of the skull. He lays some stress upon the fact that the subarachnoid fluid moves to the skull opposite to the side exposed to the motion, so that a part of the brain is thrown against the wall denuded of fluid.

Riese believes that seasickness is a purely functional disturbance dependent upon changes in the statics of the body. He describes the disturbances as occurring through a reflex arch consisting of an afferent path from the peripheral organ to the static center, and an efferent path which he assumes to be the vagus nerve. Persons, and especially old people, with decreased vagotonus are relatively immune to the condition.

Just what the vestibular apparatus of the internal ear has to do with seasickness has not been determined. The function of the semicircular canals as organs of equilibrium is unquestioned and it is fair to assume that the rapid and marked changes in the circulation of endolymph in the canals is a factor in the disease. And while the exact pathogenesis of seasickness cannot be explained wholly on this basis it does seem clear that the one essential factor constantly present is disturbance of equilibrium. This may be dependent upon some single factor but the weight of evidence points toward a combination of factors, among which kinetic changes, cerebral anemia and disturbances of the centers of equilibrium in the cerebellum and vestibular apparatus are the most prominent.

Certain classes of individuals enjoy partial or complete immunity from seasickness. The disease is rare among infants and uncommon under the age of three. Pincussen reported a case in a 10 months old infant who vomited shortly after nursing whenever the sea was rough and never when the sea was calm. Rosenbach has observed infants who were not on mother's milk and who were perfectly happy and well when the sea was quiet, but who when the sea became rough appeared apathetic and seemed in genuine distress. Old people, persons who have had suppuration of the inner ear, and to a less degree the blind have partial immunity. The susceptibility of children increases from the age at which they learn to walk up to adult life, although the disease cannot be said to be common below the age of puberty.

The **symptoms** of seasickness in infants and young children are mild, and consist of disturbed digestion with vomiting and constipation; drawn facies; and alternate stages of apathy and restlessness. The complaints of older children are the characteristic symptoms of adults modified by the natural resistance of the child.

Symptoms arising from the *central nervous system* are: headache, which is usually frontal; vertigo; asthenia; insomnia; mental depression; and prostration. *Alimentary* symptoms include: anorexia, accompanied with a disgust of food; ptyalism; nausea, from the odor of the ship, the sea, or of food; vomiting, first of food, then mucus, and in extreme cases emesis of bile or even blood; and constipation, which in most cases is persistent and obstinate. *Circulatory* findings consist of: lowered blood pressure; and rapid, small, feeble pulse, with diminished resistance.

Secondary symptoms of minor importance are: pain, postorbital, and in the muscles of the neck; pale, cold and clammy skin; and oliguria, which in some instances amounts to almost total suppression. Children commonly void only 200 to 300 c.c. in 24 hours. The specific gravity is increased to 1.030 or more. Acetone is frequently found, and in severe cases diacetic acid. There is no albumin, and sugar and cellular elements are not found. Temperature variations are not uncommon and amount in most cases to a moderate reduction.

Among the **complications** of seasickness may be mentioned premature labor, decreased secretion of breast milk, disturbances of nutrition, and hernia. In older children hemoptysis and cardiac weakness have been observed.

Since infants and children are relatively immune from seasickness, diseases simulating it should be carefully excluded. A diagnosis of seasickness in a very young infant should be made, therefore, only after careful exclusion of other diseases. Breast-fed infants frequently suffer from digestive disturbances, especially when the mother is ill; and artificially fed infants suffering from dyspepsia give practically the same clinical picture of anorexia and general indisposition as obtains in seasickness. In older children many diseases begin with vomiting, as is the case in scarlet fever, measles, meningitis, poliomyelitis, chicken pox, and diphtheria. The importance of making a correct differential diagnosis cannot be overestimated because of the danger of an epidemic of contagious diseases. This danger is increased when living conditions aboard ship are unhygienic, and especially in the closely crowded compartments of the steerage.

The **prognosis** depends upon individual predisposition to changes of equilibrium and upon the intensity of the motions of the ship. The prognosis is better in the rolling motion of the ship around the longitudinal axis than it is in the pitching motion around the horizontal axis. There is rarely any danger to life except from the rarer complications. Sudden shock as from trauma or pronounced psychic impressions as from fire are said to have cured seasickness, but this effect is only temporary, and the probabilities of recurrence of symptoms in susceptible individuals are not permanently altered by such phenomena. Occasionally one attack of seasickness produces so-called immunity from subsequent attacks. This is noticeable among children who are ill on first crossing the sea but who on return voyage remain free from symptoms. The prognosis for ultimate adjustment of the body mechanism to the ship's motion is unfavorable in very severe cases. Individuals of this group may never be able to make a sea voyage without the discomforts attending the disease.

Treatment should begin with prophylaxis. Hogg, who had many years of practical experience in dealing with cases of seasickness, insisted that treatment should begin at least 48 hours before sailing, with a diet consisting of very moderate quantities of plain and nutritious food. During the first 24 hours on shipboard, food should be taken in small amounts, and liquids and fruit avoided.

Children predisposed to kinetic disturbances should travel on the larger steamers, and when possible should remain near the center of the boat where the ship's motions are at a minimum. Odors from the kitchen and the engine room are to be avoided. A great deal of time should be spent on deck in the open air. Exercise aids in stimulating all body functions and is especially valuable in preventing the constipation which so frequently is the forerunner of seasickness. Some form of diversion should be provided for nervous children, and in this, deck games are of great value.

Active treatment should begin with the first sign of seasickness. The patient is to be put in bed and allowed small quantities of nutritious food, and should be kept in bed until nausea has disappeared. Babies retain thick cereal gruels better than fluids, and older children may be given in addition small quantities of chicken, dry toast or zweiback, and thick soups. Children who vomit persistently will often retain sticks of glucose candy when everything else is vomited. The candy stick is sucked and very slowly taken into the stomach. It is highly nutritious and is of great value in combating acidosis. Enemata of 4 per cent. glucose solution may be given for infants and children when vomiting is severe. Subcutaneous injections of normal salt solution are to be given when there is great loss of fluid, or when the normal turgor is decreased.

Alcohol in the form of wine or brandy is well tolerated by children and is a beneficial stimulant. The use of morphin is recommended by some authors but should be given only in very cautious doses to children. In certain instances improvement has followed minute doses of atropin (gr. $1/1000$ to $1/500$) given to a child of three to five years, or half this amount for younger patients.

It is not advisable to physic babies and young children. Taken by mouth cathartics irritate the entire alimentary tract and are unnecessary. Suppositories or enemata should be used to promote a daily bowel movement.

Antipyrin and phenacetin in small doses are sometimes helpful. Children requiring sedatives may be given sodium bromid, or chloral hydrate per rectum. The dose of the former for a child of five years is gr. v, and of the latter gr. ii. Following an attack of vomiting small pieces of ice may be swallowed. An ice bag applied to the abdomen to decrease the blood supply and hot compresses to the head to counteract cerebral anemia ameliorate the symptoms.

After all these means have been tried one turns to Rey for this advice: "fermez les yeux et prenez patience" (close your eyes and be patient).

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CHAPTER XXVII

BERIBERI

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Etiology.—During the many years that beriberi has been recognized clinically, there have been many theories concerning its etiology. One of the earliest theories advanced by numerous observers was that beriberi is caused by some deficiency in the food. Thus Van Leent in 1867 concluded that the principal cause of beriberi is to be found in a diet which is too uniform, insufficient in quantity and poor in quality.

Unfortunately Van Leent lived in a period when theories were based solely upon epidemiological evidence, which is often suggestive, seldom conclusive, and open to several interpretations. Experimental evidence at that time was entirely lacking. Many believed that the disease was infectious, partly because it was seasonal in its occurrence and partly because outbreaks often occurred which closely resembled the epidemics of an acute infectious disease. With the development of the science of bacteriology a number of investigators sought ot discover a causal organism: The technique employed was often extremely crude. Blood cultures, for instance, were made by sticking the finger and collecting the drops of blood for culture. Of course organisms were found, but no two investigators found the same organisms, and beriberi could not be produced by any of the organisms found. In fact, all attempts made in later years, with proper technique, to obtain causal organisms from the blood of patients suffering from beriberi have failed, as have other investigations to discover a causal organism, and for this reason the infection theory has been given up as totally without foundation.

A considerable number of observers believed that the cause of beriberi is to be found in an intoxication of some kind, assuming, usually, that the toxin is produced in rice and other foods as the result of the action of hypothetical organisms. Although a few observers still adhere to this theory the accepted opinion at the present time is that beriberi is caused by faulty metabolism resulting from a deficiency of certain accessory food substances that have been called vitamins. This opinion is based chiefly upon experimental evidence. Fraser and Stanton, in a carefully controlled human feeding experiment, demonstrated that beriberi can be produced experimentally in men by too exclusive feeding on overmilled or polished rice. This experiment was repeated later by Strong and Crowell[2] with substantially the same result. In 1897 Eijkman described a polyneuritis developing in fowls fed on an exclusive diet of polished rice. He recognized this disease as one very similar to beriberi in man, and found moreover that the addition of rice polishings to the diet would prevent the disease. The work of Eijkman led to a long series of investigations in all parts of the world. It was soon found that the protective substance or substances could be extracted from the rice polishings by both aqueous and alcoholic solvents. In 1911 Funk[3] described a crystalline base obtained from this extract of rice polishings which in the course of several hours completely cured pigeons suffering from polyneuritis that had been induced by a diet of polished rice. Funk gave to this substance the name vitamin.

Numerous other investigators have succeeded in confirming this observation, although no one has obtained the essential substance in a state of chemical purity or in sufficient amount to make an accurate chemical analysis. The reason for this failure is that for the most part the essential vitamin undergoes chemical changes during the process required to isolate it, as the result of which it loses its curative properties. Williams[4] has shown that synthetic preparations having similar curative properties, such as certain of the hydroxypyridines, display a remarkable isomerism, so that the curative form may at ordinary temperature undergo a rearrangement of its atoms resulting in a different crystalline structure and in the formation of a physiologically inert substance. It is probable that the vitamins occurring in rice polishings undergo similar changes during chemical manipulation.

In brief it may be stated that while little is known regarding the exact chemical identity of the vitamin as it occurs in foods, the fact that such substances exist, and that their deprivation results in the production of beriberi may be considered to be proved.

Practical applications of this theory are numerous and have resulted in the eradication of beriberi from various institutions or groups of men. Thus the Filipino Scouts formerly suffered greatly from beriberi, having about 500 cases a year among some 5000 men. A high-grade polished rice formed the bulk of their diet. An undermilled rice containing the external layers of the grain in which the protective substance is found was substituted, with the result that beriberi disappeared immediately and has never reappeared. Since that time undermilled rice has always been supplied. Beriberi was similarly eradicated from all civil government institutions in the Philippines. Similar results, with which every tropical sanitarian should be familiar, have been obtained in all parts of the world, though it is impracticable to quote them all here.

It should not be supposed, however, that beriberi occurs only on a rice diet. It has been shown that a number of other foodstuffs are either relatively or absolutely deficient in the beriberi preventing vitamin. Highly milled white wheat flour and various carbohydrate foods, such as tapioca, sago, and the various sugars, are quite as deficient in vitamin as rice and when used too exclusively in the diet will produce beriberi in men or fowls with great certainty. It has also been found that sterilization destroys this vitamin in most foodstuffs, so that some foods that have been canned are likewise deficient. On the other hand it has been found that whole rice or whole wheat, beans, barley, and yeast contain relatively large amounts of this vitamin, so that the use of these foods in sufficient amount will surely prevent the disease. For a further discussion of the etiology of beriberi the reader is referred to other publications on the etiology of this subject.

Historical.—For many years it was generally supposed that beriberi did not affect nursing infants. Baelz who had considerable experience in Japan, said that children never have this disease until the second dentition. However, in all Oriental countries where the native population subsist largely upon rice, and where beriberi is prevalent, the infant mortality is extremely high. This did not surprise any European who knew the conditions under which these children lived, but beriberi was not suggested seriously as an important cause of infant mortality in these countries until 1898, when Hirota described the disease in infants and reported 30 cases at a meeting of the Medical Society of Tokyo. It is said that for a number of years previously, articles had appeared in Japanese medical journals describing beriberi of nursing infants, but Miura states that none of these articles presented sufficient proof that the disease was true beriberi, and being published in Japanese, they attracted little attention outside of Japan. Hirota, however, writing in German, presented a clear description of the disease in infants, and pointed out that the mother who nursed her baby almost invariably suffered from beriberi, and also that the infant, although unresponsive to ordinary medical treatment, usually recovered at once when removed from the mother's breast and given artificial food. One year later, M. Miura[7] published the result of four necropsies on infants who had died with the clinical diagnosis of infantile beriberi. All four infants had hypertrophy of the right ventricle without valvular disease or any other condition to account for the enlargement, from which Miura concluded that this was typical of beriberi.

For many years native physicians in the Philippines had recognized that the infant mortality was exceedingly high, and that this mortality was largely due to a disease known under various native names such as Taon or Taol. Naturally this name meant nothing to American physicians, and in reporting such cases to the board of health, since an official diagnosis had to be found, many of these cases were reported as "convulsions." But in 1904 Dr. Manuel Guerrero reported 103 cases of Taon and arrived at the conclusion that it was the same disease described by Hirota as infantile beriberi. This led to a continuous series of investigations to determine the truth or falsity of this belief. McLaughlin and Andrews[8] performed necropsies on 219 infants dying under one year of age, and of this number 124, or 56.6 per cent., were found to have died of a condition which these investigators called infantile beriberi, and they pointed out the possibility that this disease might be the cause of the high infant mortality. Albert[9] likewise pointed out that while in Berlin only 9 per cent. of the infant mortality occurred among the breast fed infants, in the Philippines 65 per cent. of the infant mortality occurred among such children, and believed that this remarkable fact was due to the prevalence of infantile beriberi among nursing infants in the Philippines. Andrews[10] in 1912 made a clinical study of both the mother and the infant suffering from beriberi, followed by a necropsy of the infant when it died. He also succeeded in producing experimental beriberi in puppies that were fed entirely on the milk from the mothers whose children had just died. These puppies developed the same enlarged heart that is found with such regularity among children that have died of beriberi. In the same year Chamberlain and Vedder[11] showed that cases of infantile beriberi could be cured in a most remarkable way by the administration of an extract of rice polishings, even though the infants continued to nurse the mother and were therefore still receiving the food which was deficient. This extract of rice polishings was the same preparation that these authors had already found effective in preventing the development of polyneuritis in fowls fed on polished rice, and from which Vedder and Williams[12] later succeeded in recovering a crystalline substance capable of promptly curing fowls suffering from polyneuritis. As most of the infants who developed well-marked symptoms of beriberi died, and as the recovery following the administration of this extract was so rapid, resembling the rapidity with which a child suffering from scurvy recovers when administered orange juice, it was evident that the remedy was undoubtedly effective. Moreover, it afforded strong evidence regarding the etiology of the disease. It had previously been very generally assumed that the infant was intoxicated with some substance derived from the mother's milk, and that to insure recovery it was necessary to discontinue the use of this food. But since with the administration of this extract the infant recovered while still at the breast, it seemed most probable that the milk was not toxic but it was merely deficient in the essential vitamin which could be supplied in large amount in the extract of rice polishings.

In 1912 the Philippine legislature appointed a committee to investigate the causes of the high infantile mortality in the Philippines and adopt measures that would tend to decrease this rate. Early in 1914 this committee turned in its report, which contained the following paragraph: "In their importance as mortality and morbidity influences upon adults and children the following conditions take rank in the order mentioned: Beriberi, tuberculosis, diseases of the respiratory system, gastro-intestinal diseases, diseases of the nervous system, social diseases."

While there may be some difference of opinion as to the exact order of importance of these various diseases, especially since it would seem that social or venereal diseases are of greater importance than is indicated by this report, yet it will be seen that at least infantile beriberi is to be regarded as a most important cause of the excessive infant mortality in the Philippines. Partly as the result of this investigation, the Philippine legislature has for several years appropriated funds for the preparation of the extract of rice polishings and its distribution to the children suffering from infantile beriberi. The preparation of the extract was entrusted to the Bureau of Science in Manila, which has been most successful in this work. The report to the legislature by "La Liga Nacional Filipina para la Proteccion de la Primera Infancia" for the year 1915–1916 with regard to the manufacture and distribution of the extract of rice polishings states that 4967 bottles of extract, each containing 50 c.c., were furnished, of which 4649 bottles were used with excellent results in the treatment of 423 infants suffering from beriberi in Manila. This work, however, has had little effect in reducing the infant mortality of that city. During 1915 there were 1057 deaths from infantile beriberi or 36.8 per cent. of the total mortality of infants less than one year of age. The report concludes with a recommendation that this work be extended and combined with an educational campaign in order that mothers may learn of the efficacy of this remedy for infantile beriberi. While this endeavor is laudable, the remedy, in spite of its great success is nevertheless only a method of treating the disease after it has appeared. There is in fact a far simpler method of eradicating beriberi, to which I shall refer under the heading of prophylaxis.

Definition.—Infantile beriberi is an acute or subacute disease affecting infants who are nursing mothers suffering from beriberi. In a considerable percentage of cases the symptoms of beriberi in the mother may be very slight, and in a small percentage may be absent altogether. The disease is characterized by cardiac hypertrophy, generalized edema, chronic passive congestion of the liver, kidneys and spleen, and punctate subpleural hemorrhages, together with a certain degree of peripheral neuritis. The clinical picture is that of cardiac disturbance, with dyspnea, gastro-intestinal derangements, edema, oliguria, and aphonia. In children, as in adults, beriberi is caused by the deficiency of certain vitamins in the food, i.e., in the milk from a mother who is suffering from a similar deficiency. It is important to remember that there is no essential difference between beriberi in adults and beriberi in infants, except for the fact that as the infant neither walks nor talks, it is impossible to elicit the various motor, sensory, and subjective symptoms that are obtained from the adult.

Pathology.—The majority of deaths occur in the acute cases. Postmortem examination usually shows an infant healthy in appearance who seems to be well nourished. Some edema is generally present. The skin of the legs may pit on pressure, and appear anemic and pale. On cutting into the body there is often a free flow of fluid blood, and it is noticed that the subcutaneous fat is moist or edematous. This condition leads one to suspect that there is not so much fat as was supposed, and that the well-nourished appearance may have been due in part to the edema. On opening the chest, it is at once seen that the heart is enlarged. The pericardium is frequently filled with a clear fluid having a characteristic yellowish-green color. The cardiac enlargement is seen to be chiefly of the right ventricle which may measure from 5 to 7 mm. in thickness, whereas the left ventricle, normally the larger, measures only 3 to 5 mm. The weight of the heart in nine of Andrews' cases averaged 34.1 gm., while the weight of the heart of the average healthy infant of the same age does not exceed 20 gm. The musculature of the right heart is coarse and firm and forms the greater part of the organ, so that the left ventricle may even be excluded from the apex.

There may be considerable pleural fluid, which if present resembles the fluid found in the pericardium. The lungs themselves are practically normal except for the fact that there may be a few petechial hemorrhages beneath the visceral pleura. A certan amount of fluid may often be expressed but it is probably the result of pulmonary edema which develops shortly before death.

On opening the peritoneal cavity an increased amount of peritoneal fluid is usually found, again of a yellowish-green color. The liver is of a dark reddish-brown color, is congested and may sometimes show a slight nutmeg appearance. Sections may also show slight albuminous degeneration, and in some cases there is considerable hemorrhage beneath the capsule. The spleen is also markedly congested, and microscopic examination may show that hemorrhages have occurred into the splenic tissue. The kidneys are congested, and on cut section show a very moist surface from which a considerable amount of blood oozes forth, and sometimes a few subcapsular hemorrhages may be found. Aside from this congestion the kidneys appear practically normal, but some sections show albuminous degeneration. On opening the skull the meninges probably will also be found congested and edematous, with possibly some increase in the amount of cerebrospinal fluid, but no other abnormality.

It will thus be seen that there are four points of special interest in these findings: The dilated and hypertrophied right heart, the congestion of the viscera, the general tendency to anasarca and effusions of a peculiar greenish fluid, and last the absence of findings sufficient to account for death. No reasonable explanation has ever been given for the production of the peculiar cardiac hypertrophy in beriberi. Although there has been much speculation concerning both this and the production of the anasarca, and while it is evident that these conditions are produced as the result of the dietary deficiency, we are still in the dark as to the physiological action of this vitamin and the exact manner in which its deprivation brings about these changes.

If the nerves are examined by the Marchi method, degeneration will be found in certain of the fibers, but the extent of this degeneration apparently bears no relation to the intensity of the other changes. In some cases this nerve degeneration may be quite extensive, while the heart is only moderately enlarged, whereas other cases in whom the heart is greatly enlarged and the tendency to anasarca pronounced, the degeneration of the nerves may be slight and in some cases may be difficult to detect at all.

Symptomatology.—In children, as in adults, the disease manifests itself in a variety of types. Guerrero, who has had much experience, classifies the cases as incipient, aphonic-acute, aphonic-chronic, and fulminating. Albert in a recent paper read before the Manila Medical Society described a pseudo-meningitic form of the disease characterized by choreic movements of the limbs which lasted for several weeks followed by convulsions, twitching of the facial muscles, strabismus, slight rigidity of the neck and slight fever. Lumbar puncture gave negative results. *X*-ray examination in these cases showed the enlargement of the right heart, the characteristic lesion of the disease. But such cases, even though they may occur, are quite rare. It is far simpler to divide the cases of beriberi into two types:(1) The acute, pernicious or cardialgic type, lasting from several hours to several days and characterized by intense and incessant crying and sudden death; and (2) the chronic type which lasts for several weeks.

Acute Beriberi in Infants.—This is very abrupt in its onset. A young infant, usually from one to four months of age who has always been healthy previously, is suddenly seized with paroxysms of pain. The child straightens out its body and becomes rigid. He cries constantly. The face becomes cyanosed, the pulse is small and rapid, and the heart flutters. The heart is enlarged to the right in all these cases, and thise serves as a diagnostic point. If the attack passes off, it is usually succeeded by others of greater or less length, and the child may die in a few minutes or in several hours. Death is sudden and apparently the result of cardiac failure.

On questioning the mother it is found that the child has been breast fed and has never had other food; frequently the mother will state that she has had several children die in the same way. It often happens that mothers having lost several children through this disease, and realizing that their deaths were due to breast milk, will try to raise the next one on artificial feedings. An examination of the mother will usually elicit mild symptoms of beriberi, such as inability to walk far, tenderness of the calf muscles, anesthesia over the shins and diminished patellar reflexes. It is recognized that this history must appear rather sketchy to one who has never seen the disease, but it is usually all that is obtainable. And having seen a number of children die in this manner, and having found in all of them at postmortem the lesions already described, one is convinced that however trifling the symptoms, the condition is not only very real but very fatal.

Chronic infantile beriberi presents a somewhat different picture. The first symptoms are usually disturbances of the gastro-intestinal tract, particularly constipation and vomiting. The constipation is obstinate, and only yields temporarily to medicinal treatment. The vomiting is often repeated several times during the day at irregular intervals that bear no relation to the time of nursing. The ejecta consist entirely of milk with a little mucus. After a few days the child becomes restless and fretful, particularly at night, so that a good night's sleep is rarely obtained. These symptoms persist in spite of medicinal treatment. Gradually the child becomes pale and his skin assumes a waxy tinge that is very characteristic in the brown skin of the native child. The restlessness is very noticeable and is accompanied by a feeble whining cry. A diminution in the amount of urine now occurs, presenting a symptom that the mothers all notice. In severe cases only a few drops of water may be passed in the 24 hours, so that the number of napkins used in a day is greatly diminished. (In regard to napkins, it may be said that the Filipino baby is often without such impedimenta.) We have then a noticeable oliguria, associated with which there may be a tendency to anasarca, the lower extremities being usually affected. This may lead to a suspicion of nephritis, which is not confirmed by the examination of the urine. This dropsy may be absent entirely. Later the voice disappears or at least is strangely altered. This condition is termed aphonia. A plaintive whine remains, but it is quite different from the normal voice of the child. It has always been thought that this aphonia was caused by degenerative changes in the pneumogastric nerve. It is a symptom which occurs rarely in adults suffering with beriberi, but is rather common in infants. There are usually some cardiac symptoms. The impulse of the heart may be rather violent, and the apex beat displaced downward. Percussion or x-ray examination shows the heart, especially the right ventricle, to be greatly enlarged. The heart sounds may be rapid and the second pulmonic sound markedly accentuated. There are usually no murmurs. If the cardiac condition is severe, it is accompanied with marked dyspnea which recurs in paroxysms of variable duration. Albuminuria is infrequent. There are no symptoms referable to the nerves of the limbs such as one would expect to find in beriberi, but it must be remembered that the patients, as a general rule, are infants of three months of age or less, so that the nervous system cannot be tested with much satisfaction. In all of these cases, if the mother continues to nurse the infant, the condition almost always ends in death, which is usually sudden, resulting apparently from the cardiac dilatation and failure which is so common in beriberi. If a change is made to artificial feeding, a certain proportion of these children can be saved, and this proportion would undoubtedly be larger were it not for the ignorance of the native mothers as to proper methods of artificial feeding and the poverty that prevents them from obtaining suitable milk and ice.

The age at which beriberi occurs in infants is of importance. The disease sometimes appears soon after birth. The great majority of cases, however, occur before three months of age. After four months they are rare, and they are practically unknown in infants over a year of age. With regard to the age at which beriberi attacks children, it may be noted that during the first few months of life that the infant subsists entirely on maternal milk. In later months they often receive some food which may help to make up the existing deficiency. The depletion period of beriberi in the human has been shown to be about 90 days; that is, if men subsist exclusively upon a diet of beriberi-producing rice, about 90 days will elapse before the first appearance of the disease. It is evident, therefore, that in children subsisting on a milk that is deficient, the majority of cases of beriberi should appear 90 days after such feeding has begun. As a matter of fact, the disease occurs in children in a shorter period than this, probably because the growing child is more susceptible to this deficiency than the adult. Moreover, since these infants are born of mothers who themselves are already suffering from the effects of the deficiency of the vitamins that prevent the disease, it may be assumed that they are incapable of supplying the fetus with a sufficient supply of these substances, so that the child is already suffering from this deficiency when it is born. When a child is nursed by a mother who is also deficient in vitamins, it is evident that he will develop beriberi often in less than the 90 days, which has been observed to be the shortest depletion period in adults.

The relation of this disease to infantile scurvy is of some interest. It is obviously not the same disease, not only because the clinical symptoms and pathological anatomy are different in the two conditions, but because fruit juices which produce such remarkable cures in scurvy have no effect in alleviating infantile berberi. There are however some clinical resemblances. Hess[13] has pointed out that an enlargement of the heart and more especially of the right heart may be found in some of the cases suffering from infantile scurvy, while degeneration of the nerves has also been demonstrated in certain cases of scurvy in adults. It seems evident that beriberi and scurvy are closely related diseases, both being caused by a deficiency in vitamins. It seems also evident that there are several of these vitamins, and that the deficiency of the beriberi-preventing vitamin causes beriberi while the deficiency of the scurvy vitamin produces scurvy. If both were missing, the patient should suffer from a combination of the two diseases, and this has indeed been the case in many outbreaks of ship beriberi. Until more evidence has been secured as to the exact chemical nature of the vitamins, and the role that they play in the metabolism of the body, such explanations as the above are to be borne in mind as purely speculative hypotheses.

Naturally attempts have been made to study the milk of mothers of children suffering from beriberi in order to determine the nature of the deficiency. In brief it may be said that many of these mothers were supplying the infant with milk which was normal so far as the ordinary analysis for fats, carbohydrates, proteids, inorganic salts and phosphorus were concerned, and that no light has been thrown on the problem by analysis. It may be assumed that since these infants are cured by the extract of rice polishings, that the deficiency in the mother's milk is made good by the vitamin that has been proved to be present in this extract.

Treatment.—From the first it has been recognized that medicines are useless in beriberi. Hirota and subsequent observers, in accordance with the theory that the child was receiving a toxin in the mother's milk, have treated this condition by removing the child from the breast and substituting artificial feeding. But this treatment is far from satisfactory among poor and ignorant native mothers. It is difficult enough to train mothers to prepare milk properly in the United States, but it is impossible in the Philippines, and I presume the same is true in Japan and other Oriental countries. The treatment introduced by Chamberlain and Vedder in the Philippines has given continued satisfaction in the hands of other observers, and may be regarded as practically a specific for the disease which will save practically all cases not already moribund. This treatment consists in giving the child an extract of rice polishings in such dosage that it will receive the protective substances from about 100 gm. of rice polishings daily. This treatment should be continued for several weeks, during which the child should continue to nurse its mother. This is all the treatment that is necessary and it has the great advantage of enabling the child to continue to nurse. Guerrero[14] has recently reported a series of 349 infants suffering from infantile beriberi, including all stages of the disease and a few practically moribund cases, all of whom were treated by this method. Of these infants, 234 or 67 per cent. were cured, 33 or 9.4 per cent. were improved, and only 7.1 per cent. died. The remainder of the series were lost to further observation. As the disease when untreated is almost uniformly fatal, these results speak very highly for the success of this treatment.

Method of Preparing the Extract of Rice Polishings.—Rice polishings in the Philippines and in the United States are quite different. In the Philippines all the substances removed from the grain except the hulls are blended together. In the United States the various parts of the grain are kept separate. What is called rice polish is only the starchy part removed from the grain in the final polishing. The aleurone layer which contains the protective substances is mostly contained in the part called rice meal, although a considerable portion is in the exterior layer removed, which is called rice bran. The rice polishings to which reference is made in this article is the Philippine product consisting of a mixture of all the material removed from the rice. The polishings are sifted, weighed and extracted with 90 per cent. alcohol. During extraction the alcohol becomes deeply tinged with green by the fat which is dissolved. This extraction may be repeated with several portions of alcohol. The alcohol so obtained is filtered and evaporated on a water-bath at not higher than 80°C. An electric fan facilitates the evaporation. When the alcohol is entirely evaporated, the residue is poured into a separating funnel and allowed to stand for about an hour, at the end of which the liquid will separate into two layers. The upper and larger portion, which is deep green, is the fat; the lower layer is brown in color, of syrupy consistency and contains the vitamins that have been extracted, together with sugar, resins, and other substances. As the vitamins were found to be soluble in water, this lower layer is carefully drawn off and diluted with water to about twice its bulk, whereupon a heavy precipitate of resins and other similar substances soluble in alcohol but not in water is formed. This precipitate is filtered off, and the clear brownish fluid is the extract of rice polishings ready for use. The preparation is sweet and has a very agreeable taste, which infants like. It may easily be administered from a medicine dropper.

Prophylaxis.—The prevention of disease is always more satisfactory than its treatment. The prophylaxis of beriberi in infants is the same as for other forms of the disease, since it will not occur in infants if the mothers receive food containing a proper supply of vitamins. Thus, although beriberi has occurred in the United States in certain jails, asylums, and other institutions where the diet has been one-sided and deficient, it does not occur among the population of this country as a whole. In fact, no case of an infant affected by this disease has ever been reported. Practical experience has shown that beriberi can be eradicated, providing a beriberi-preventing diet can be substituted for the diet responsible for the production of the disease.

In Oriental countries the diet that is responsible for the production of beriberi is polished or overmilled rice from which the external layers have been completely removed. Rice is the staple foodstuff in these countries, and because of custom and economic considerations it will always form the bulk of the diet of the masses of the native population. The majority of these natives who suffer from beriberi are both too poor and too ignorant to supplement their diet with the various articles that will prevent the disease, such as beans, barley, and fresh meat. Therefore, the only hope of eradicating beriberi from these countries lies in the substitution of an unpolished or undermilled rice for the polished rice that is in general use. This unpolished or undermilled rice retains the external layers of the grain which contain the beriberi preventing vitamins, and it has been shown conclusively that beriberi-never occurs when this undermilled rice is used.

Unfortunately this simple recommendation will never be carried out without some form of governmental action, because people as a general rule, and especially native races, will not change their accustomed food habits except by compulsion. Let us suppose that some serious disease were found to be due to the practice of eating white bread, and that it could be entirely prevented by eating whole wheat bread. It is highly improbable that any except the most intelligent would make the desired change. At the first suggestion of such a change, all the millers would rise up in arms, because this entails a change in the usual methods of milling wheat. With the usual conservatism of business men, they insist that the present methods are the best methods, they refuse to believe that any flour that they produce could possibly be deficient in anything, and they absolutely refuse to consider a change.

In the Orient, the same reluctance is encountered when a change in the method of milling rice is suggested. In the Philippines it was suggested by Dr. Victor G. Heiser, then Director of Health, that a tax be placed on the highly milled rice which produced beriberi, making the price of this rice prohibitive for the poor, thus compelling them to purchase the undermilled and beriberi-preventing rice, the price of which, instead of advancing, might even drop. A bill to this effect was introduced into the legislature in 1911 and again in 1912, but for various reasons it failed to pass. In the meantime the legislature has appropriated funds for the treatment of infants suffering from beriberi. It seems quite apparent that the attempt to use this successful method of treatment as a preventive measure will prove a failure, in spite of the fact that the extract of rice polishings is practically a specific, because of the impossibility of reaching the large number of cases. One can hardly fail to conclude that instead of endeavoring to meet the situation by treatment, the legislature should take steps to pass laws that will tax the polished rice, and thereby compel the use of the undermilled and beriberi-preventing rice by the poor who are the main sufferers from the disease.

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CHAPTER XXVIII

ACIDOSIS

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Acidosis is a process which results in the depletion of the fixed alkalies of the body. During the course of normal metabolism considerable amounts of acid are produced, such as carbonic, phosphoric and sulphuric as well as certain organic acids. Under normal conditions the body is able to eliminate these acids or to neutralize them with ammonia so that there is no depletion of the reserve of fixed alkali in the blood and tissues. If, however, acids are produced in excessive amounts, if the mechanism of elimination and neutralization fails, if there is an excessive intake of inorganic acid or insufficient intake of bases the alkali reserve may be drawn upon and when this occurs the condition of acidosis may be said to be present.

Though some authors still follow Naunyn* and consider as acidosis any condition which results in the excessive production of acids of the acetone series, it is the custom of the majority to speak of acidosis only when the mechanism for the defense of the body against acids is brought into play to an excessive degree and the reserve alkali depleted to a greater or less extent.

The Defenses of the Body against Acid.—Acidosis may be recognized by excessive activity of the defenses of the body against acid, hence an understanding of these processes is essential. It may be assumed that the blood plasma differs but slightly in composition from the tissue juices throughout the body and that any change in the acid base equilibrium of the tissue juices will be reflected in the composition of the blood plasma. A consideration of the acid base equilibrium in the blood plasma enables us to understand the processes taking place in the body as a whole.

The constituents of the plasma which are most important in preserving the normal alkalinity are sodium bicarbonate, proteins and inorganic phosphates. The sodium bicarbonate is relatively of much greater importance than either of the other two substances. For practical purposes the plasma may be considered as a solution of sodium bicarbonate of a strength of approximately 0.3 per cent. A solution of sodium bicarbonate of this strength is definitely alkalin in reaction, but if carbon dioxide is passed into the solution the reaction becomes less alkalin and may ultimately become actually acid, but a considerable amount of carbon dioxide may be taken up in solution without very greatly changing the reaction. As the blood plasma circulates through the body, carbon dioxide is poured into it from the tissues as the result of their metabolic activity. This changes slightly the reaction of the plasma rendering it less alkalin although during life the plasma probably never becomes definitely acid. The change in reaction, however, occurring as the result of an increase in dissolved carbon dioxid, is sufficient to stimulate the respiratory center and this stimulation produces an activity of the respiratory muscles. The pulmonary ventilation thus brought about leads to elimination of the carbon dioxide, but as more carbon dioxide is always being poured into the plasma the respiratory center is once more stimulated after a short interval of time. When excessive amounts of carbon dioxide are produced in the body, as, for example, after exercise, greater stimulation of the respiratory center occurs and the respirations become deeper and more rapid and thus the removal of the excess of carbon dioxide is accomplished. There is no depletion of the fixed alkalies.

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* Naunyn, "Diabetes Mellitus," Vienna (1906).

When non-volatile acids such as beta-oxybutyric, aceto-acetic, or lactic are poured into the plasma, the first effect is to neutralize part of the sodium bicarbonate with liberation of carbon dioxide. The effect is the same as if acid were added to a solution of sodium bicarbonate in a test tube. The carbon dioxide liberated decreases the alkalinity of the plasma and consequently there occurs a stimulation of the respiratory center. The increased respiratory activity tends to remove the excess of carbon dioxide. Whether this can be completely accomplished or not depends upon the vital capacity of the lungs. Even if the excess of carbon dioxide is removed so that the alkalinity of the plasma returns to normal, the plasma still does not contain the same amount of bicarbonate that it normally should, as some of the bicarbonate has been neutralized by the non-volatile acid. When this plasma containing a diminished amount of sodium bicarbonate again passes through the tissues and takes up carbon dioxide, its reaction becomes less alkalin with the same amount of dissolved carbon dioxide as formerly when it contained its full bicarbonate content. Roughly speaking, if the bicarbonate of the plasma is reduced one-half, only half as much carbonic acid can be carried in solution as formerly if the reaction is to remain the same as before. By doubling the pulmonary ventilation the dissolved carbonic acid of the plasma may be diminished about one-half, and if the bicarbonate of the blood plasma is also reduced one-half the reaction of the blood leaving the lungs will be the same as under normal conditions, but until the bicarbonate which has been depleted has been restored, excessive pulmonary ventilation must continue. It is for this reason that deep breathing or *hyperpnea* is one of the striking clinical evidences of acidosis. If pulmonary ventilation is doubled without there being at the same time an increased production of carbon dioxide in the body, the percentage or "tension" of carbon dioxide in the air leaving the lungs will be diminished one-half. For this reason the tension of carbon dioxide in the alveolar air may be taken as an indication of the degree of acidosis present. When the tension of carbon dioxide in the alveolar air is diminished one-half it is, generally speaking, evidence of the fact that the bicarbonate reserve of the blood is diminished one-half and the pulmonary ventilation has been doubled.

Increased pulmonary ventilation serves as a temporary measure to prevent excessive changes in the reaction of the blood and tissues, but it cannot prevent a depletion of the reserve alkalinity. To replenish the depleted bicarbonate the body brings into play other protective mechanisms.

With a slight change in the reaction of the blood, there is an increased production of ammonia in the body. The ammonia being a base combines with acid radicals and is quickly excreted in the form of the ammonium salts of these acids. This serves to protect the sodium bicarbonate from depletion. An excessive amount of ammonium salts in the urine is evidence that this mechanism has been called into play, hence it is taken as one of the laboratory evidences of acidosis.

When the bicarbonate of the blood plasma is diminished, this may be restored to some extent as the blood passes through the kidney due to the specific activity of this organ in excreting acid. The excretion of an acid urine necessarily leads to an increase in the alkalinity of the blood passing through the kidney. Only a small amount of acid is excreted in the free state in the urine, but a very considerable amount in the form of acid phosphate. The plasma, as has already been mentioned, contains some inorganic phosphate. This is chiefly in the form of alkalin phosphate. Alkalin phosphate in solution in the plasma passing through the kidney may be excreted in the form of acid phosphate with the result that the bicarbonate of the plasma is increased. The following formula shows how this is brought about:

$$\mathrm{Na_2HPO_4 + H_2O + CO_2 = \overset{\text{Excreted in Urine}}{NaH_2PO_4} \quad + \overset{\text{Remains in Blood}}{HNaCO_3}}$$

When the alkalies of the body are depleted, the kidney responds by excreting a relatively large proportion of acid as compared with alkaline phosphate in the urine. The urine is normally acid but in the condition of acidosis it becomes still more acid. There is, however, a limit to this process of acid excretion and the kidney is incapable of excreting a urine the acidity of which is very greatly in excess of the normal acidity. When a urine is alkaline when passed or when its acidity does not exceed the normal this may be taken as evidence of the fact that acidosis is not present, but an increase in the acidity of the urine above normal limits is not always reliable evidence of the presence of acidosis.

The Clinical Diagnosis of Acidosis.—The most distinctive clinical evidence of acidosis is the characteristic "air hunger." The breathing of acidosis is *deep*, the increase in rate is less marked. The breathing is very different from the shallow, rapid respirations of pneumonia or the stridulous, labored breathing of obstruction. The respirations of acidosis are pauseless and although individual respirations may vary in depth, in general the excursions of the abdomen and thorax are nearly the same with succeeding respirations. The most striking feature is the amplitude of the respirations and the distinct effort with which they are accomplished. The respirations are heaving, the chest rises and falls with each respiration and often the accessory muscles are brought into play. There is no cyanosis except in the presence of cardio-respiratory disease.

A cherry-red coloration of the lips is occasionally observed, but is of little diagnostic value. In severe acidosis, especially that brought about by an excessive production of acetone bodies, drowsiness may be a symptom. The odor of the breath is of significance only in those cases of acidosis due to the acetone bodies. Vomiting is not a symptom of acidosis. In the condition of recurrent vomiting of children both vomiting and acidosis are present, but the vomiting is not the result of the acidosis nor is it the cause. The vomiting occurs often before any appreciable acidosis is present. Furthermore, in the more severe types of acidosis, such as, for example, that occurring in the course of diabetes, vomiting is not a prominent symptom.

The Laboratory Diagnosis of Acidosis.—Several relatively simple laboratory methods for the detection and quantitative estimation of the degree of acidosis are available. The following are the methods best adapted to clinical use:

1. *The Determination of the Carbon Dioxide Tension in the Alveolar Air.*—This determination may be carried out in a few minutes' time at the bedside by the use of Marriott's method.* The method is applicable to infants as well as older children and adults. The alveolar air is collected by having the patient rebreathe air from a rubber bag. The sample of air thus obtained is analyzed by bubbling it through a standard bicarbonate solution containing an indicator and comparing the color with that of standard tubes. For the details of the method see the original article referred to or the directions accompanying the standard sets.

In normal children at rest the carbon dioxide tension in the alveolar air varies from 40 to 45 mm. Tensions between 30 and 35 mm. are indicative of a mild degree of acidosis. When the tension is as low as 20 mm. the individual may be considered in a critical condition. In coma associated with acidosis the tension may be as low as 8 or 10 mm. In infants the tension of carbon dioxide is from 3 to 5 mm. lower than in older children.

Care is necessary in interpreting the results as conditions other than acidosis may affect the carbon dioxide tension, for example, anything which affects the respiratory center may so change the respirations as to lead to variations in the carbon dioxide of the alveolar air. The respiratory center may be affected by such drugs as caffein which lowers the threshold of stimulation and causes the center to respond to a less degree of alkali reduction of the plasma than under normal circumstances. Certain intracranial lesions have been known to have a similar effect.

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* Marriott, Jour. Am. Med. Assn., lxvi (1916), p. 1594. The apparatus for this determination may be obtained from Hynson, Westcott and Dunning, Baltimore.

In diabetes especially after treatment with large amounts of alkali and during periods of partial starvation the alveolar carbon dioxide tension may be lower than would be expected from the bicarbonate reserve of the blood. This seems to be due to alterations in the excitability of the respiratory center.

Changes in the pulmonary epithelium capable of preventing the air in the lungs coming in equilibrium with the blood in the pulmonary capillaries would of necessity affect the composition of the alveolar air. We are, therefore, not justified in drawing far-reaching conclusions regarding acidosis merely from the composition of the alveolar air in patients suffering from severe pulmonary affections.

In the majority of cases the determination of the carbon dioxide tension of the alveolar air is sufficient evidence on which to base a diagnosis of acidosis, but where possible it is advisable to check the determination by an examination of the blood to determine the actual bicarbonate content.

2. *Determination of the Bicarbonate Reserve of the Plasma.*—This may best be accomplished by the method of Van Slyke.* This method depends upon the principle that if acid is added to a solution of sodium bicarbonate the amount of carbon dioxide liberated is proportionate to the amount of bicarbonate present in the solution. For the determination 1 c.c. of serum or plasma is usually used. Certain precautions must be observed in the collection of the blood. The patient should be at rest for an hour previous to drawing the blood. If an arm vein is used and it is necessary to apply a tourniquet this should be released as soon as the needle enters the vein and a few seconds allowed for the blood to pass before any is withdrawn. In the case of infants blood may be obtained from the superior longitudinal sinus. Blood obtained by cupping is likely to yield results which are less accurate on account of the escape of carbon dioxide. The blood, however obtained, must be protected from exposure to the air and all unnecessary agitation must be avoided. Coagulation may be prevented by a few crystals of neutral potassium oxalate. The tube should be centrifuged as soon as possible after the blood has been drawn and the clear plasma pipetted off for the determination. All glassware used should either be paraffined or should be made of "Pyrex" or "Nonsol" glass, as ordinary glass yields appreciable amounts of alkali to solutions.

Before determining the bicarbonate content of the plasma it should be saturated with carbon dioxide at normal alveolar tension. This may be most readily accomplished by collecting alveolar air in a rubber bag as for the determinations of alveolar carbon dioxide tension and bubbling this air for one minute through the plasma contained in a test tube. A bottle of glass beads should be interposed between the bag and the tube to condense the moisture from the breath.

* Van Slyke, Jour. Biol. Chem., xxx (1917), p. 347.

Before bubbling the air through, a fine glass rod moistened with octyl (caprylic) alcohol is touched to the surface of the plasma. This serves to prevent foaming. The determination of the carbon dioxide content of the plasma thus prepared is carried out in the special apparatus.* When analyzed by this method the plasmas of normal adults and older children contain from 50 to 78 volumes per cent. of combined carbon dioxide. In infants the normal values average about 10 per cent. lower. Plasma bicarbonate contents lower than 50 volumes per cent. in adults or 40 volumes per cent. in infants are indicative of acidosis. The results if multiplied by 0.7 approximate alveolar carbon dioxide tensions in millimeters determined according to the method previously described and have the same significance.

3. *Alkali Tolerance Test.*—This method was first described by Sellards and has been extensively used. It depends upon the fact that when the bicarbonate content of the plasma is within normal limits the administration of a small amount of sodium bicarbonate by mouth brings about an alteration of the reaction of the urine. The excess of bicarbonate is excreted and the normally acid urine becomes amphoteric or alkalin. When there is acidosis the bicarbonate of the plasma as well as that of all of the body tissues is diminished. This must be replenished before the bicarbonate administered will be excreted by the kidney. For this reason the amount of soda that must be given to produce an amphoteric or alkalin urine is a measure of the depletion of the bicarbonate reserve of the body. With infants, 1 or 2 gm. of sodium bicarbonate is sufficient to lead to the excretion of a urine which is alkaline to litmus. Five grams of sodium bicarbonate will accomplish the same result in older children and adults. When acidosis is present very large amounts of soda, up to 10 times this amount, may be required to bring about a change of the reaction of the urine.

The method amounts essentially to a titration of the body with alkali to determine the amount necessary to bring it back to normal conditions. The method has the advantage of a double utility. It enables one to make the diagnosis of acidosis and at the same time to cure the condition, for when the urine has become alkalin to litmus the acidosis is overcome, temporarily at least.

Litmus paper is not the best indicator to use in making this test as an alkaline urine is evidence that the acidosis has been more than overcome since the urine under normal conditions is not alkaline to litmus but has a reaction slightly to the acid side. The indicator that we have found most satisfactory has been cresol purple (dibromcresol-sulphone-phthalein).† This indicator changes color at just about the average reaction of the normal urine. The indicator is made up in alcoholic solution and a drop added to a small amount of freshly passed urine. If the color changes to violet or purple it is evidence that acidosis is not present; if it remains green acidosis may or may not be present; alkali should then be administered by mouth until the urine just causes a purple coloration of the indicator. Under normal conditions very small amounts of bicarbonate by mouth are sufficient to effect this change.

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* This apparatus is obtainable from the Emil Greiner Co., 55 Fulton St., New York. For further details the reader is referred to Van Slyke's original article or to the directions accompanying the instruments.

† Obtained from Hynson-Westcott and Dunning, Baltimore.

The three methods described above are those most available for the clinician. Other methods which have been used and which are satisfactory are the serum test of Sellards,* the indicator-dialysis method for the determination of the reserve alkali,† and the determination of the combining power of hemoglobin for oxygen.‡

All of the methods above referred to are applicable for the detection of acidosis of any type and from any cause. There are other methods which are applicable only to certain types of acidosis.

Methods Applicable Only for the Detection of Certain Types of Acidosis. *Acetone Bodies in the Urine.*—In acidosis due to an over-production of acetone bodies, B-oxybutyric acid, aceto-acetic acid and acetone appear in the urine in sufficient amounts to be detected by qualitative tests, but such qualitative tests are insufficient evidence on which to base the diagnosis of acidosis. Acetonuria is exceedingly common with sick children. It is almost regularly present in febrile diseases and in any condition in which temporary inanition or starvation occurs. In many of these instances, although sufficient acetone bodies are present to give a qualitative test in the urine, the amounts are insufficient to lead to any detectable reduction in the alkali reserve of the body. From acetonuria alone the diagnosis of acidosis should never be made though when the amount in the urine is very large it should inspire a careful examination for additional evidence of acidosis. One must not lose sight of the fact that acidosis due to the acetone bodies is only one of several types commonly occurring and that severe and fatal acidosis frequently occurs when there is no over-production of acetone bodies and then they cannot be detected in the urine.

Ammonia Coefficient.—In acidosis due to increased production of organic acids or the ingestion of strong mineral acids the excretion of ammonium salts in the urine is greater than under normal conditions. There are types of acidosis, however, notably that occurring in the course of nephritis, in which there is no increase in the excretion of ammonium salts. A high excretion of ammonia is only presumptive evidence of acidosis. A normal excretion of ammonia does not rule out the condition.

Clinical Types of Acidosis. *Acidosis Due to the Acetone Bodies.*—When fat is metabolized in the body under such conditions that only a relatively small amount of carbohydrate is simultaneously metabolized, an incomplete breakdown of the fatty acid molecules results. The katabolism of fatty acids stops at the stage of the acetone bodies; acetone, oxybutyric and aceto-acetic acid. The latter two of these substances are acids capable of bringing about the condition of acidosis.

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* Sellards, Johns Hopkins Hospital Bull., xxv (1914), p. 101.

† Marriott, Arch. Int. Med., xvii (1916), p. 840.

‡ Barcroft, The Respiratory Function of the Blood, Cambridge (1914). For the details of these methods see the original papers referred to.

During starvation, when the body has used up to a large extent its carbohydrate stores, and when the fat depots are drawn upon, considerable amounts of the acetone bodies may be excreted in the urine. Acidosis of a moderate degree may be present though probably never of a severe enough degree to threaten life.

During the course of fever, from any cause, there may be an increase in the basal metabolism and this together with the simultaneous reduction in the intake of food often results in an increased production of the acetone bodies. Acetonuria is indeed almost a constant accompaniment of fever. In some of the infectious diseases an over-production of acetone bodies occurs to such an extent that it can hardly be explained merely on the basis of the fever. It is possible that in these diseases the toxic agent exerts a direct effect upon the organs concerned in the metabolism of fat and carbohydrate. Among the diseases which are likely to be complicated by an acetone body acidosis of a greater or less degree are dysentery, pneumonia, acute rhino-pharyngeal infections, scarlet fever, measles and typhoid fever.

In diabetes an increased production of the acetone bodies occurs as a result of the failure of the body to utilize carbohydrates. For a detailed discussion of the acidosis of diabetes the reader is referred to the article on diabetes in this volume.

Following surgical operations, especially in children, a marked over-production of the acetone bodies may occur and this not infrequently leads to a condition of acidosis. It is not yet entirely clear just how this acidosis is brought about. The acidosis which occurs is not entirely due to the acetone bodies as the amount of these is not always sufficient to account for the degree of acidosis observed.

In the condition known as cyclic or recurrent vomiting severe and even fatal acidosis may occur although, in the majority of instances, even when the attack is prolonged, acidosis plays a very small if any part in the picture of the disease. The acetone bodies are occasionally found in the urine in very large amounts. At times they are to be detected a few hours after the onset of vomiting, so early that their presence at these times can hardly be accounted for on the theory of starvation. Acetone may be detected in the breath of these patients and can be determined quantitatively in the blood. The accumulation of acetone bodies does not account for the vomiting, nor, if acidosis is present, will treatment of the condition by alkali therapy necessarily bring the vomiting to a conclusion. The serious complication of acidosis may be overcome by alkalies but the cause of the attack of vomiting is not removed.

Infants, at the end of the first and in the second year, may suffer from attacks of *recurrent acidosis* due to the over-production of the acetone bodies. With these infants the vomiting may play a very subordinate rôle. There may, in addition, be slight diarrhea but it is the manifestations of acidosis that dominate the picture. In pediatric practice this type of acidosis has not sufficiently been recognized. Unless vigorous treatment is instituted the attacks are likely to prove fatal. It is probable that this condition of recurrent acidosis bears a close relationship to recurrent vomiting.

Acidosis Due to Lactic Acid.—Lactic acid is produced in excess during severe exercise, during convulsions, in all asphyxial conditions, in surgical shock, in phosphorus poisoning and in acute yellow atrophy of the liver. This increased production or accumulation of lactic acid in some instances leads to the condition of acidosis.

Acidosis Due to Unknown Organic Acids.—A distinct acidosis is occasionally observed in lobar pneumonia. The acidosis is of a more severe degree than can be accounted for by the relatively small amounts of the acetone bodies present. According to Palmer* the acidosis is due to an organic acid of unknown composition which is excreted in the urine.

The Acidosis of Cardio-respiratory Disease.—Patients with certain congenital malformations of the heart and those suffering from cardiac decompensation may develop acidosis as a result of the failure of the poorly circulating blood to transport carbon dioxide effectively from the tissues to the lungs. The acidosis in such cases is due in part to carbon dioxide and in part to lactic acid. The latter substance is produced in those tissues, which as the result of cardio-respiratory failure, receive an insufficient oxygen supply.

In patients suffering from bronchial asthma and in those with extensive pulmonary involvement or obstruction in the respiratory passages, the elimination of carbon dioxide from the lungs is greatly interfered with and an acidosis may develop. There is increased stimulation of the respiratory center with resulting hyperpnea. The carbon dioxide tension in the alveolar air is diminished, but on account of the fact that acidosis is due to carbon dioxide the reserve alkalinity of the body may not be depleted. Some lactic acid is, however, usually produced in these conditions as the result of asphyxia and this is capable of depleting the alkali reserve.

The Acidosis of Renal Origin.—The ability of the kidneys to excrete acid, especially acid phosphate, is one of the chief defensive mechanisms of the body against acids. Failure of this mechanism, even without increased production of acids in the body, results in acidosis. Occasionally, in nephritis, acidosis occurs and it has been shown† that when acidosis is present there is an accumulation of unexcreted phosphate in the blood plasma. There is no evidence that any abnormal acid is produced. In these cases of renal acidosis hyperpnea is present, the carbon dioxide tension in the alveolar air is lower than normal, the alkali reserve of the blood is lowered and there is an increased "alkali tolerance." No acetone bodies are to be detected in the urine and the ammonia excretion is normal or even diminished. Failure to produce ammonia to neutralize the retained acid is an additional factor in the production of acidosis of nephritis,* and represents a weakening of another of the body's defenses against acid.

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* Palmer, Jour. Exp. Med., xxvi (1917), p. 495.

† Marriott and Howland, Arch. Int. Med., xviii (1916), p. 708.

Acidosis Occurring with Diarrhea.—Czerny† made the observation that malnourished infants suffering from diarrhea develop a type of respiration much like that of animals poisoned by acids and suggested that these infants were suffering from a form of acid intoxication. Later Czerny and Keller‡ found that the urines of these infants frequently contained a relatively large amount of ammonia nitrogen. They considered this finding additional evidence that acidosis was present. They were unable to find any abnormal acids present in the urine. Steinitz,§ in a series of metabolic experiments on infants suffering from diarrhea, was able to show that there was a negative sodium balance as a result of an excessive elimination of sodium salts by the bowel. He advanced the theory that the acidosis of these infants was the result of loss of alkali by the bowel. He failed, however, to take into consideration the fact that acid radicals are lost as well. Indeed, more recent work by Holt, Courtney and Fales‖ and of Shohl¶ has shown that the loss of acid radicals may be relatively greater than that of bases. The conception of a "relative acidosis," therefore, rests on a very insecure foundation.

Howland and Marriott** have brought forward definite evidence that in the severe, watery diarrheas of infants (cholera infantum, intoxication, etc.) a severe degree of acidosis may result. These findings have been confirmed by Schloss†† and by Ylppö.‡‡ The acidosis is not present in all cases of infantile diarrhea or even in all severe cases, but when present it is a very serious and usually fatal complication. In these infants with acidosis the carbon dioxide tension of the alveolar air is low, the alkali tolerance is much increased, the alkali reserve of the blood plasma is diminished and there is an alteration in the hemoglobin dissociation curve. The characteristic deep breathing is present.

The cause of the acidosis occurring in severe infantile diarrhea is not always plain. The acidosis is not the result of an excessive production of the acetone bodies although these are at times moderately increased; at other times in the presence of severe acidosis they are within normal limits. The acidosis is in part explained on the same basis as the acidosis of advanced renal disease, that is to say, by a failure of the kidney to excrete acid phosphate. We have been able to demonstrate an excess of phosphate in the blood of some of these infants during the stage of acidosis. The failure of the kidneys to eliminate acid in these cases is the result of a functional incapacity due to the fact that so much fluid has been lost from the body by other channels that the urine secretion becomes almost impossible. A striking feature of the clinical picture in these infants is the very scanty secretion of urine. An infant may secrete only an ounce or two of urine in the course of a day.

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* Henderson and Palmer, Arch. Int. Med., xvi (1915), p. 109.

† Czerny, Jahrb. f. Kinderh., xlv (1897), p. 271.

‡ Czerny and Keller, Jahrb. f. Kinderh., xlv (1897), p. 274.

§ Steinitz, Jahrb. f. Kinderh., lviii (1903), p. 689.

‖ Holt, Courtney and Fales, Amer. Jour. Dis. Ch., ix (1915), p. 213.

¶ Shohl, Unpublished work.

** Howland and Marriott, Amer. Jour. Dis. Ch., xi (1916), p. 309.

†† Schloss, Amer. Jour. Dis. Ch., xiii (1917), p. 218.

‡‡ Ylppö, Zeitschr. f. Kinderh., xiv (1916).

In not all of these infants can an excess of phosphate in the serum be demonstrated, so that it is unreasonable to suppose that it is the only factor in the production of the acidosis. Marriott* has advanced the theory that the acidosis is due to the accumulation in the blood and tissues of acids resulting from incomplete oxidation. The cause of the incomplete oxidation is attributable to the greatly diminished volume flow of the blood which has been shown to occur in these infants. The volume flow of the blood is decreased because of the reduction in blood volume and this in turn depends upon the concentration of the blood through loss of fluid. The acidosis often disappears after the administration of a sufficient amount of fluid to restore the blood volume to the normal amount and to allow for the resumption of renal activity. A condition of acidosis has been experimentally produced in animals by reducing the blood volume and volume flow. This acidosis also disappears when the blood volume is restored.

Acidosis Due to the Ingestion of Acids.—Mineral acids taken by mouth in sufficient amount may lead to the condition of acidosis but this is a rare cause. Certain articles of food contain inorganic constituents in which the excess of acids over bases is relatively great but it is doubtful if a diet rich in such articles of food administered to a healthy person would ever lead to the production of a significant degree of acidosis.

Acidosis Due to Poisons.—Acidosis has been demonstrated in the case of persons suffering from poisoning by a number of substances. Methyl alcohol leads to the condition probably on account of the production of formic acid (Krol,† Harrop‡). Emetine§ in large doses has been known to lead to the condition in human beings. Phosphorus, arsenic and a number of war gases are also capable of producing acidosis.

Acidosis in Burns and in Surgical Shock.—Patients suffering from severe surface burns may develop the type of respiration characteristic of acidosis and Blackfan‖ has brought forward definite evidence that acidosis is actually present in some of these patients. In surgical shock, acidosis is often present as has been repeatedly demonstrated. The acidosis is to be considered a complication rather than a cause of the shock as has been advocated by some. The acidosis in both burns and surgical shock is not due entirely to the acetone bodies and probably not to failure of the kidney to excrete acid. There is in both conditions a diminished volume flow of the blood and this may well lead to the acidosis. The acidosis sometimes seen following hemorrhage is likely of similar origin.

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* Marriott, Amer. Jour. Dis. Ch., xx (1920).

† Krol, Arch. Exp. Path. u. Pharm., lxxii (1913), p. 442.

‡ Harrop, Jour. Am. Med. Assn., lxxiv (1920), p. 25.

§ Levy and Rowntree, Arch. Int. Med., xvii (1916), p. 420.

‖ Blackfan, unpublished.

Prognosis.—The prognosis in cases of acidosis depends upon the cause as well as upon the severity of the acidosis and also upon the underlying condition. To cure the acidosis is not necessarily to cure the disease of which the acidosis is a complication. In general, it may be said that acidosis of the acetone body type offers the best prognosis. With diabetes the prognosis is bad on account of the incurability of the fundamental condition and because of the fact that a severe degree of acidosis is indicative of advanced diabetes. In attacks of recurrent vomiting and with the acetone body acidosis of small children, the prognosis is good if the condition is recognized early and vigorous treatment adopted. The acidosis occurring in the course of renal disease is serious even if the acidosis itself is of a moderate degree. The outlook is far better in acute nephritis than in the chronic types. When acidosis occurs in the course of diarrhea it adds a very dangerous complication to an already serious condition. When acidosis of a severe grade occurs in such cases recovery is unusual.

Treatment of Acidosis.—The treatment of acidosis is to prevent the production of more acids, whenever possible; to replenish the depleted alkali reserve, and to eliminate the acids and their salts.

In diabetes the production of acids may be best prevented by strict adherence to the proper dietary regime (for details of the treatment see article on "Diabetes").

With recurrent vomiting glucose solutions intravenously are theoretically indicated, especially if the blood sugar shows a tendency to fall. With the development of acetone body acidosis, glucose should be given in all cases except those due to diabetes. The glucose may be given by mouth or by rectum or intravenously. If given by mouth it may be administered in a 5 or 10 per cent. solution every two to four hours in quantities depending upon the age of the child and the tolerance of the gastro-intestinal tract. When administered by rectum a 5 per cent. solution is to be employed which may be injected in large amounts at long intervals or better by the drip method if this is tolerated. By this latter method we have seen as much as a liter of 5 per cent. glucose solution absorbed within 24 hours' time. For intravenous use a 5 or 10 per cent. solution of glucose may be employed in amounts up to 20 c.c. per kg. of body-weight. The solution should be given very slowly or otherwise glycosuria is likely to result.

In the acidosis accompanying cyclic vomiting the diet of the child for some time after an attack should contain very little fat and not an excessive amount of protein. It is very desirable in these children that their bowels should be kept open. A good cathartic for this purpose is sodium phosphate.

Alkali Therapy.—In all types of acidosis, if a considerable depletion of the alkali reserve has occurred, alkalies are indicated. Sodium bicarbonate is usually used for this purpose. The amount of alkali needed to restore the alkali reserve may be easily calculated. The blood and tissue fluids contain approximately 3 gm. of sodium bicarbonate per kg. As approximately 70 per cent. of the body-weight is made up of such fluids, the total amount of bicarbonate in the body is approximately 2 gm. per kg. of body-weight. If the blood bicarbonate or the alveolar carbon dioxide tension is diminished one-half, then it is necessary to administer 1 gm. of bicarbonate per kg. of body-weight to restore normal conditions. This may be expressed in the form of a formula as follows: $\frac{40 - T}{40} \times 2\,W =$ grams of sodium bicarbonate needed; or, $\frac{60 - V}{60} \times 2\,W =$ grams of sodium bicarbonate needed. Either of these formulæ may be used depending whether the degree of acidosis is measured by a determination of the alveolar carbon dioxide tension or by the bicarbonate reserve of the blood plasma. In the formulas $T =$ alveolar CO_2 tension in millimeters, $V =$ volume per cent. CO_2 in plasma as determined by the Van Slyke method, $W =$ body-weight in kilograms.

The amount of bicarbonate, as calculated by this method, is the amount necessary to return the alkali reserve of the body to normal. It does not allow for the continued production of acids nor does it take into account the possible elimination of these acids.

In applying this formula to cases of acidosis it will be seen that the amounts of sodium bicarbonate needed are often large. The failure of alkali therapy in acidosis is often due to the fact that an insufficient amount of alkali is given. When the deficit of sodium bicarbonate in the body amounts to 50 gm. or more the administration of 5 or 10 gm. of course has but little effect.

Knowing the amount of alkali necessary, this may be administered in one of several ways. It may be given by mouth in doses of from 1 to 4 gm. at intervals of two to four hours. It should be given in solution and well diluted. It may be made somewhat more palatable, without at the same time reducing its efficiency, by the addition of a small amount of citric acid or lemon juice to cause effervescence. Appropriate flavoring may be added if desired by the patient. In some cases of acidosis vomiting renders the administration of bicarbonate by mouth impossible. Under such circumstances, if the acidosis is of a severe grade (alveolar carbon dioxide tension 25 mm. or lower) the intravenous method of administration should be used. A 4 per cent. solution is commonly employed for this purpose. It is prepared by adding sodium bicarbonate to freshly distilled and freshly sterilized water. The solution must not be subsequently boiled or heated much above body temperature as the bicarbonate breaks up with the liberation of carbon dioxide and the formation of sodium carbonate. A solution prepared as described does not require further sterilization as a solution of sodium bicarbonate of this strength is sterile. The amount of the solution to be injected depends upon the severity of the acidosis and the size of the patient. From 15 to 20 c.c. per kg. of body-weight is the maximum amount which can be safely injected at one time without throwing too great a burden on the circulation. The injections should invariably be given slowly. The bicarbonate may be administered in this way several times a day.

The administration of bicarbonate should be continued until the alkali reserve of the body has returned to approximately normal limits. In severe acidosis of the acetone body type the alveolar carbon dioxide tension may for a period remain low even after the alkali reserve of the blood has returned to normal. The respirations may also continue to be deep. This is presumably the result of an hyperirritable respiratory center. When it is not feasible to determine the bicarbonate reserve of the blood plasma a very simple clinical test may be applied which indicates accurately when a sufficient amount of alkali has been given. This test consists in adding a drop of cresol purple to a fresh specimen of urine. If the color remains green, more alkali is necessary; if it changes to any shade of red or purple, a sufficient amount of alkali has been given.

There is little danger to the patient in the administration of an excess of sodium bicarbonate. Edema may occur, but in our experience it is not a dangerous sequel. In young children and in nephritics typical tetany sometimes accompanied by convulsions may occur following the administration of large doses of sodium bicarbonate, but with the cessation of alkali administration a return to normal conditions rapidly takes place. When massive doses of sodium bicarbonate are to be administered there are theoretical as well as practical reasons for administering magnesium sulphate subcutaneously as a prophylactic against tetany. This may be given in 8 per cent. solution in doses up to 0.2 gm. per kg. of body-weight.

Water Administration.—In all cases of acidosis, whether mild or severe and whether alkali is administered or not, water is urgently required for the elimination of acids and their salts. Water is, in fact, more necessary even than alkali and in many instances it is all that is necessary. The water is to be given by mouth in as large amounts as can be borne. Vomiting frequently makes oral administration difficult or impossible. A certain amount of fluid can, however, often be retained when it is given almost continuously and in small amounts at a time. For this purpose a small catheter is introduced through the nose into the lower part of the esophagus but not into the stomach. Salt solution or tap water is allowed to drop into this at the rate of 10 to 20 drops a minute. When all methods of oral administration fail, the choice is then between rectal, intravenous, subcutaneous or intraperitoneal administration of normal saline solution. If diarrhea is present or the rectum is intolerant, rectal administration is out of the question. Intravenous injection is unsatisfactory for the reason that it must frequently be repeated as a relatively small amount of fluid can be given at one time. Larger amounts may be administered by hypodermoclysis, but even by this method the amount which it is possible to give is often insufficient especially in the case of young infants.

We have found the intraperitoneal injection of fluid to be far the most satisfactory in these cases. This is especially true of those infants suffering from diarrhea and the accompanying desiccation. The water deficit of these infants is frequently very great, amounting to several hundred cubic centimeters, and the administration of fluid by this means is frequently life saving. Either normal saline or Ringer's solution may be used. The injection is made either in the median line below the umbilicus or outside the left rectus muscle at about the height of the umbilicus. A short needle with a short bevel is used. The fluid warmed to body temperature should be injected slowly and with all aseptic precautions. As much as 200 to 350 c.c. may be given at a single injection to a small infant. The injection may be repeated several times a day if the fluid is absorbed. There is no resulting discomfort and the fluid is absorbed gradually without throwing any strain on the circulation. The clinical indications that a sufficient amount of fluid has been given are a resumption of secretion of urine in normal amounts and a return of the skin to its normal elasticity.

With the methods of treatment outlined above, it is usually possible to cure temporarily the acidosis, but unless the underlying cause is found and removed the condition will recur. Even though the acidosis is successfully cured the patient may die as the result of the original disease of which the acidosis is a complication, or if a severe acidosis has been unrecognized and untreated for any considerable period of time the body may have become so damaged that a continuance of the life processes is impossible even though the acidosis has been corrected.

The successful treatment of acidosis depends upon an early appreciation of its presence, a correct diagnosis of the type and discriminating judgment in the employment of means sufficient to overcome the disturbance.

CHAPTER XXIX

OBESITY

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Obesity may be briefly described as the excessive accumulation or deposit of fat in the body. While the condition is less common in childhood than in later life, it is nevertheless not infrequent even at an early age. Occurring most frequently at or near the age of puberty, extreme degrees of the affection are occasionally met with even in the first year of life.* The general characteristics of the disease appear to be in the main quite similar to those met with in adults, although actual metabolic studies in children have been very few. However, a brief discussion of the metabolic factors involved is quite essential to a proper understanding of the etiology and therapy.

The relative energy requirements of a child are considerably in excess of those in adult life. In the full-grown adult all that is required is a maintenance diet which will enable the body functions to be carried on properly plus sufficient calories to cover the energy (heat) used up in work. In the child, however, additional requirements must be made for growth. Children's dietaries, therefore, must be made quite liberal in order to provide for an increased muscular activity in the child, and to supply a certain amount of material to be added to the body in the form of new tissue in addition to that which is oxidized to support metabolism. The amount of food required for healthy maintenance per unit of body-weight decreases from infancy to old age.

Recent investigations tend to show that the "basal metabolism," that is, the metabolism while at complete muscular rest, is proportionate to the surface area of the body and that even in obesity this is not altered. Benedict and Talbot, however, believe that in infants the relation between the basal metabolism and the body surface is not uniform, and that when newborn infants are included, there may be a variation of over 100 per cent.† They feel that the active mass of protoplasmic tissue alone determines the fundamental metabolism. While this is doubtless true, unfortunately we are unable to measure the amount of this active protoplasmic tissue. Studies on the basal metabolism of infants and young children are difficult because of the inability of keeping them still long enough. Benedict and Talbot, however, state that in general, normal babies, not newborns, weighing the average for their age, have a basal metabolism of, roughly, 50 to 65 calories per kg. of body-weight irrespective of age. This is the lowest amount of energy on which a baby can maintain its body functions. They add that the metabolism may be increased from 40 to 100 per cent. above the basal metabolism by the change from complete muscular repose to active exercise. Crying, for example, will raise the amount of energy expended 40 per cent. or more. Babies that are over-weight have a basal metabolism of between 40 and 52 calories per kg. Fat infants are usually quieter than babies which have developed their muscles, and will, therefore, gain more weight on a low energy quotient (calories per kg.) than the average active child. During the first weeks of life the metabolism is very low, after which it rises rapidly, being highest in the first six months of life, and dropping slightly in the next half year. At the end of the first year it is still 50 per cent. above the adult level. Sherman states that children of normal size, development, and activity, will require about as follows:

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* The writer has seen an infant seven months old weighing 34 lb., and another 16 months old weighing 46 lb. The "Dictionnaire Encyclopédique" cites the following amazing cases of obesity in childhood, with the author citing the case: Th. Bartholin, a 10-year-old child weighing 200 lb.; Kaestner, one of four years weighing 82 lb.; Benzenberg, one of four years weighing 137 lb.; Percy and Laurent a child weighing 13 lb. at birth, 42 lb. at six months, 150 lb. at four years, and 450 lb. at 20 years.

† The basal metabolism in infants is usually computed after a meal instead of after a 14 to 18 hour fast, as in adults.

1 to 2 years	1000 to 1200 calories per day
2 to 5 years	1200 to 1500 calories per day
6 to 9 years	1400 to 2000 calories per day
10 to 13 years (girls)	1800 to 2400 calories per day
10 to 13 years (boys)	2300 to 3000 calories per day
14 to 17 years (girls)	2200 to 2600 calories per day
14 to 17 years (boys)	2800 to 4000 calories per day

Greatly increased muscular activity would, of course, raise the caloric requirements, and as children of the same age vary considerably in size, it is well to have some idea of the food requirements per unit of body-weight. To quote from Sherman, again, the following approximate values may be followed:

Under 1 year	100 calories per kilogram	(45 calories per pound)
1 to 2 years	100 to 90 calories per kilogram	(45 to 40 calories per pound)
2 to 5 years	90 to 80 calories per kilogram	(40 to 36 calories per pound)
6 to 9 years	80 to 70 calories per kilogram	(36 to 32 calories per pound)
10 to 13 years	75 to 60 calories per kilogram	(34 to 27 calories per pound)
14 to 17 years	65 to 50 calories per kilogram	(30 to 22 calories per pound)

Next we must bear in mind the theoretical considerations which go to make up a well balanced diet, for it is of especial importance in childhood to see that the protein needs of the growing organism are fully covered, even though dietetic measures are instituted aiming at a reduction or limitation of excess deposits of fat. The protein needs of a child constitute about 9 to 13 per cent. of the total calories, and may be stated as about a minimum of 1.5 gm., or better between 2 and 3 gm. protein per kilogram of body-weight. While there is no absolute data as to the proper proportions of fat and carbohydrate, it seems likely that for very young children the food value of the fats should be somewhat higher than that of the carbohydrates, and for those somewhat older, the calories should be about the same for each. Fats and carbohydrates are of almost equal importance as protein sparers. In preparing a restricted diet for a child, therefore, there should be at least the minimum protein requirement of 1.5 gm. per kilogram of body-weight, while the remainder of the calories should be about equally divided between carbohydrates and fats.

Having briefly considered the basic metabolic factors which are concerned in the nutrition and growth of normal children, we may now turn to a discussion of metabolism in relation to abnormal conditions such as in obesity.

In obesity we are dealing with a state of over-nutrition, in which a deposit or storing up of fat in the subcutaneous tissues and various organs occurs either because of the ingestion of more food than the body can burn up, or because the oxidizing powers of the organism itself have been lowered. By far the greater number of cases come under the heading of so-called "exogenous obesity," in which there is a normal metabolism with an excessive supply of food; that is, more energy is furnished than the organism utilizes either in maintaining the body functions or in body labor—intake exceeds body expenditures. This excess of ingested food may be more real than apparent. The amount eaten may appear to be quite normal, but when the quality is examined it is found that the diet consists largely of food rich in fat and carbohydrates, such as candy, pastries, bread and butter, etc., with very little of the bulky foods such as vegetables and fruits, which have a low caloric value, so that the total calories represent far more than the requirement. In some cases the number of calories may be only slightly in excess of the requirement, but if this overfeeding is continued day in and day out, especially in a child who exercises insufficiently, the amount of excess food will be considerable in a year's time. In some children this excess is represented as fat deposited in various parts of the body. In determining whether or not the food ingested exceeds the body needs consideration must be taken of the child's activity. A phlegmatic, inactive child requires less food than one of the nervous, high-strung type, who is never still a moment. Obese children are usually less active than normal children, and this lessened activity easily offsets the increased work of moving a larger body. But it must be emphasized that children require more calories (probably one and one-half to two and one-half times more) per kg. than adults.

It has been customary when speaking of the obesities to refer to a small group of cases in which the disproportion between the body needs and the food intake is not apparent, and to refer to this group as of the "endogenous" type, or obesity in which there is a "constitutional predisposition," in the sense of a lowered metabolism. It has long been known that obesity often shows a strong hereditary tendency but most modern observers deny that a diminished power of burning up foodstuffs plays more than a minor rôle, if in fact it enters into consideration at all. Indeed, most observers take the view that the basal metabolism per unit of surface area is normal in obesity, except in those types of obesity due to some disturbance in one or the other of the ductless glands. It seems more likely that the cause must be sought in some disturbance of the reflex mechanism governing appetite, the feeling of satiety after the taking of food, etc. As to just what significance excessive consumption of water plays in obesity is not definitely determined, but probably the chief reason for the increased drinking of fluids by fat individuals is to have sufficient water available for the regulation of body temperature through sweating, since fat deposits interfere with heat radiation. Drinking water with meals may stimulate the appetite in certain cases, but restriction of fluids does not seem to increase the consumption of energy as was once believed.

Etiology.—We may say, therefore, that in general obesity is due to a disproportion between the food ingested and the food required for body maintenance, energy and muscular exercise. The excess food is then largely converted into fat and stored as such in the subcutaneous tissues and various organs.

Obesity occurs more frequently in female than in male children. The influence of heredity is undeniable, and in many instances the antecedents will give not only a history of obesity, but very often of gout, asthma or diabetes mellitus. In children, incidental factors such as the change from an active to a sedentary life, or diminished muscular activity, the use of alcoholics, etc., do not play nearly as important a rôle as in the adult. However, obesity occasionally follows certain infectious diseases, especially typhoid and, more rarely, certain forms of tuberculosis. In a small proportion of cases the exact etiologic factor is not readily determinable but careful search will usually reveal the fact that in most of these cases there exists a disproportion between food intake and body needs. Finally, there are types of obesity which occur in conjunction with disturbances in certain of the ductless glands. An example of this type is the well-known obesity of hypothyroidism in which there is an actual lowering of the basal metabolism, strikingly shown in cretinism. Similar disturbances in the function of the pituitary, the pineal, and the sex glands may also be followed by excessive deposits of fat. These types will be found discussed more fully in the chapters devoted to a consideration of the functions and diseases of these glands.

Pathology.—There have been very few extensive studies on the pathology of obesity in young children, but enough has been done to show that in general there is a close similarity between the picture at an early age and that seen in adult life, with the important exception that in children there is much less tendency toward degenerative changes in the heart and kidney than in adults. One sees, however, the thick layer of yellow subcutaneous fat as in adults, deposits of fat in the mesentery, in and between the muscles, around the kidneys, the heart and the pericardium. Microscopically, the liver cells are found stuffed with fat droplets and, to a lesser extent, the myocardium also. Here and there appear areas of fatty degeneration, as well as infiltration, but this is less marked than in older subjects. The kidney, too, is involved in the process, but only in the severest cases is its function impaired.

Symptoms.—The clinical picture of obesity in childhood differs somewhat from that seen in adults, especially in the more general distribution of the fat. The face is round, "moon-like," but there are not the huge jowls and pendulous double chin so frequently seen in adults. The eyes appear small and set deep in the orbits. The abdomen is smaller, proportionately, than in older individuals. The circumference of the arms and legs is usually much increased over the normal, the upper arm and thigh showing a greater degree of enlargement than the lower part of the arm and leg. There are large pads of fat at the hips, the back, the axillæ and the buttocks, while the breasts, in both sexes, are much enlarged from the subcutaneous deposit of fat in this region. The genitals appear small because their size is not increased in proportion to the neighboring tissue. There is a more or less complete disappearance of many of the bony landmarks of the body, and the flesh is soft and elastic. Deep creases and folds appear at the wrists, ankles, and neck, and often these creases as well as those in the groin are the seat of a stubborn intertrigo. Many of these children, particularly those with a tendency to eczema, have ruddy cheeks, but occasionally one sees the opposite or "anemic" type with pale, pasty-looking cheeks. In the latter group the tissues appear edematous rather than obese. Occasionally the obese subject is found to be a young girl with signs of chlorosis. Obese children are usually phlegmatic and have poor endurance partly, perhaps, because of the added muscular exertion needed to move their comparatively large bodies. Their walk is awkward, their breathing is hurried, and slight exertion increases the dyspnea out of all proportion to the degree of muscular activity. These children are inclined to sleep more than the average child, especially after meals. As a part of the necessary mechanism in regulating body temperature obese children perspire freely because of the lessened radiation of heat through the heavy layer of fat.

In childhood, serious derangement of the cardiorenal system is much rarer than in adults. Occasionally, however, a myocarditis may develop and a consequent passive congestion of the kidney, liver and spleen. In older children hemorrhoids are not uncommon. Chronic nephritis is rare. However, the presence of so much adipose tissue means more work for the heart than was intended for it, and this strain is increased by even moderate exertion. Such a condition produces a picture of dyspnea, palpitation after exercise, with a tendency toward syncope, and if the heart strain is carried to excess, sudden, acute dilatation may occur. The dyspnea may be exaggerated further by mechanical interference of adipose tissue with the fullest lung expansion and proper diaphragm action. The respiratory tract in fat individuals seems less resistant to infections also, so that acute and chronic catarrhal processes are frequently met with, followed by localized areas of emphysema. Indeed, there is apt to be a lessened resistance to infections in general in the obese.

As for the digestive organs, little need be added to what has already been said. Gastric function is usually normal. Constipation, however, often occurs, due largely to the unsuitable diet and the disinclination to exercise, but wearied abdominal muscles and perhaps, in some degree, interference with normal intestinal peristalsis, because of excessive fat deposits in the mesentery, may be contributing causes. The circulatory disturbances may lead to hemorrhoids, but again much less frequently in childhood than in later life.

Prophylaxis and Treatment.—Of paramount importance in combating obesity is prophylaxis. It is the duty of every physician who comes in contact with children showing such tendencies to warn the parents of the possible evil consequences and suggest proper steps for the control of the condition, no matter how young the child. Although mothers often take pride in the possession of an extremely fat baby, such children present a far less prepossessing appearance to the disinterested outsider, and to the pediatrician they suggest a physical state which is hardly compatible with the best of health. In infants showing a tendency towards obesity, care should be taken that the theoretical food requirements of 100 calories per kg. of body-weight are not exceeded. In breast-fed infants this can usually be accomplished by lengthening the feeding intervals to four hours, and perhaps limiting the time at the breast. The amount of food should be controlled by weighing the child before and after nursing to ascertain the exact amount of food ingested. Similarly the infant on artificial food must have his diet carefully regulated, due consideration being paid to the possibilities of the child getting food between feedings, such as sugar-water, cakes, etc., not ordered by the physician. In older children again, regardless of the caretaker's assurances to the contrary, careful examination of the diet will usually show an excess over the requirement. In the latter instance, careful diet lists must be prepared, in which proper attention is given to the caloric requirements of the average child at the same age. In the preparation of such a diet list, it must be remembered that what is aimed at is not a reduction in weight, but a nearly stationary weight until such time when the body growth will tend to restore again the normal body proportions. To attain these ends the diet lists should state specifically the kind and amount of food at each meal, preference being made for vegetables and other low calorie filling foods to produce a feeling of satiation which keeps the child happy.

Actual "reduction cures" in which loss of weight is sought are seldom advisable, and indeed frequently dangerous. Children under such regimes often become weak, pale and languid, and it may take many months for them to regain the lost bloom of their former healthy appearance. Should a reduction cure be deemed advisable in spite of these dangers, the first consideration is always to provide a diet which will amply cover the protein needs of the growing child. This can be done even in older children by ordering lean meats and other foods rich in protein.

Probably all that is needed in the average case of obesity in childhood is (1) to lessen the caloric intake, while at the same time maintaining a suitable proportion between protein, carbohydrate and fat in the food, and (2) to regulate the life of the patient so that sufficient muscular exercise is provided. Exercise must be carefully graduated at first so as not to overtax the strength of the child, and then slowly increased in proportion to the improvement in the general condition of the patient. A valuable aid in lessening the caloric intake is the cultivation of the habit of eating slowly with the ingestion of a minimal quantity of fluids at meals. The various hydrotherapeutic measures so frequently advised in the treatment of obesity in adults are scarcely necessary in childhood, but swimming forms an excellent and agreeable substitute for older children. Other exercise, such as walking, roller skating, tennis, handball, baseball, etc., may all be employed with great advantage in appropriate cases. The administration of extracts of thyroid gland is to be condemned except where there is sufficient evidence of thyroid deficiency.

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CHAPTER XXX

INFANTILE SCURVY (BARLOW'S DISEASE)

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History.—We owe the first description of infantile scurvy to Glisson, who likewise was the first to recognize rickets. Glisson's description, written in 1668, was entirely lost sight of, however, until the end of the last century. In 1859 Moeller described cases which were evidently scurvy, but which he termed "acute rickets," failing to distinguish the distinctive nature of the disorder. Soon after this, Ingerslev, Jalland, and Cheadle described cases of scurvy, the latter clearly recognizing the disease as the same which occurs in adults. In 1883 Barlow furnished anatomical proof that the disorder of infants presented the pathological changes characteristic of adult scurvy. The work of Barlow was accepted remarkably quickly in England and America, but less promptly on the continent, due to the fact that the disorder was far more prevalent in the English speaking countries. An idea of the extent of its recognition may be gained from the fact that in 1894, 106 cases were reported at a medical meeting in New York City, and that four years later a comprehensive investigation, based on 379 cases, was published by the American Pediatric Society.

Infantile scurvy has been reported from almost all parts of the world. Strange to state, in spite of the fact that adult scurvy is endemic in many parts of Russia, reports of infantile scurvy have been remarkably few from this country. Filatow, the great Russian children's specialist, declared some years ago that he knew of no case of Barlow's disease described in the Russian literature.

During the past few years, as a result of the World War, there has been a great increase of scurvy in Europe, especially in the Central Empires. Erdheim recently reported 31 autopsies on infants who had suffered from scurvy. Eric Mueller and Brandt have published accounts of scurvy in the foundling asylums of Berlin. Epstein of Prague wrote a paper with the descriptive title "On a Marked Increase of Barlow's Disease During the Years of the War 1917–1918." Vogt reported an outbreak of infantile scurvy in his hospital at Magdeburg.

PATHOLOGY

As stated, the pathology of infantile scurvy dates back of Barlow's exposition, the modern milestone in the study of the pathology of this disorder. Until recently attention has been focused exclusively on the bones, all other organs of the body being neglected. This is true of the gross, as well as of the microscopic, anatomy. Lately, however, the field has been broadened. As a result largely of the interest in experimental scurvy, the heart, endocrine glands and other organs have been subjected to close inquiry.

Gross Pathology.—The skin is pale and dotted with numerous petechiæ or echymoses of moderate size. Generally there is some edema about the ankles. The emaciation is not extreme unless the picture has been complicated by starvation or by general infection. Enlargement of the long bones, especially of the femur and tibia, may be noted, due to subperiosteal hemorrhage, and may be associated with fracture and crepitation. Another bony alteration which is readily palpable is "beading" of the ribs, the counterpart of the rachitic rosary. On incision, one of the striking phenomena is the anasarca of the soft parts. In one of the first cases with postmortem examination Barlow described this condition as follows: "The muscular walls of the thorax were pale yellow and watery, as though they had been bathed in serum." Any or all of the serous cavities may be involved in this hydrops, the order of frequency being the pericardium, pleura, peritoneum and joints, especially the knee. On opening the thorax the heart will be noted frequently as enlarged—an alteration overlooked until recently. In the article referred to above entitled "Das Barlowherz," Erdheim described enlargement of the heart, especially of the right ventricle, in 21 of the 31 necropsies of infants who died of scurvy in Vienna during the World War. In view of these protocols and of the clinical demonstration of cardiac enlargement by means of roentgenograms (Hess), and of the pathological reports in adult scurvy, enlargement of the heart must be regarded as a true scorbutic lesion.

The lungs are almost always congested. The main lesion of the gastro-intestinal tract is an intense congestion of the duodenum, frequently found in the upper part, and ulcers of the large intestine, especially where secondary infection has occurred. The liver and spleen usually are of normal size.

Microscopic Pathology.—Our knowledge of the minute pathology of the bone is far more complete and detailed than that of any other structure, as might be expected, in view of the fact that the bony resions are the only ones which are pathognomonic. One of the peculiarities of the lesion is that it involves the end of the diaphysis or rather the junction of the diaphysis and the cartilage. The bones most apt to show typical changes are the ribs; we shall, therefore, describe a section made through a costochondral junction.

The costochondral junction is generally swollen, somewhat beaded, and when cut longitudinally shows on gross examination a transverse yellowish bar, corresponding to the area of disorganization described later. Under the microscope the line of juncture is not sharp and straight as it is normally, but presents a wavy or irregular contour, the cartilage jutting into the bony end of the rib, instead of abutting in neat apposition to it. At this site is the Truemmerfeld zone, where the normal tissue is splintered and fragmented. Everything is in a state of disorder—trabeculæ of bone of various shapes and sizes lie scattered about, the cells irregularly arranged and much distorted, signs of recent hemorrhage, unrecognizable detritus. Higher magnification shows that there are few osteoblasts, a varying number of intact red cells, and occasional spindle and star-shaped connective-tissue cells.

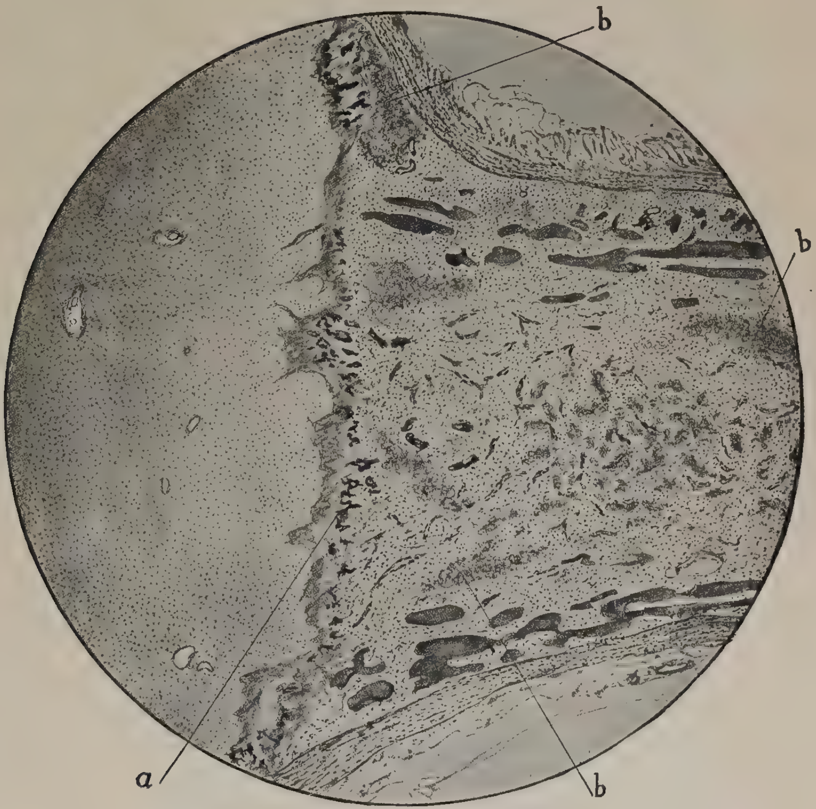

Fig. 143.—Infantile scurvy. Epiphyseal junction of lower end of femur: *a* calcified cartilage at "Truemmerfeld;" *b*, hemorrhage in the fibrous marrow (Gernest-mark). Bone cortex and trabeculæ abnormally thin. (MacCallum, "Text-book of Pathology.")

The cartilage also is not normal. Its cells do not present an orderly arrangement, the proliferating columns having disappeared in the central convex portion. This disturbance of the columnar formation of the proliferating cartilage is one of the early signs of scurvy. Below the Truemmerfeld is the Geruestmark or framework-marrow, another distinguishing lesion of scurvy. It is composed of a loosely constructed fibrillar tissue on a gelatinous-appearing groundwork of sparsely scattered cells, and bony trabeculæ which are markedly thin and weak. Here and there are hemorrhages and blood pigment, especially adjacent to the "Truemmerfeld," and the entire marrow area is incompletely filled by the thin and greatly depleted trabeculæ of the spongiosa. It is evident that the shriveled osteoblasts are not functioning normally. The osteoclasts are not present in excess. The resulting lesion is clearly the result of almost normal bone resorption with a lack of normal bone regeneration (Fig. 143).

Hemorrhage occurs in the bone as elsewhere; it is almost never lacking. It may consist of large hemorrhages in the spongiosa, or merely of scattered cells. Its most typical site is beneath the periosteum, a lesion widely known on account of its clinical significance. The inner surface of the periosteum is frequently lined with newly formed bone and with a more or less dense deposition of lime salts, which renders it readily apparent in radiographs. As a result of the weakening of the bone there is frequently a separation of the diaphysis from the epiphysis, or more correctly, a fracture or infraction of the bones. In the case of the rib this results in the displacement of the cartilage, and therefore an "angular beading" of the rib is brought about. In the course of healing a large number of giant-cells appear, blood-vessels sprout from the periosteum as well as from the bony surface, and the necrosed tissue gives place to an active formation of callus.

In addition to these changes in the bone, there are less constant alterations in some of the other organs. The muscles frequently show a diverse picture of old or recent hemorrhage. Thrombi may be found in the vessels in the neighborhood of hemorrhage. There is a difference of opinion as to whether the walls of the blood-vessels show fatty or hyaline degeneration. Hemorrhages may be found in the lungs or beneath the pleura. Although the heart is increased in size, no constant microscopic changes have been recorded in its musculature. The liver, spleen, kidneys, and lymph-nodes are the occasional sites of hemorrhage. No changes have been recorded in the adrenal glands either in the scurvy of adults or of infants. In view of the fact that changes in these organs have been described in connection with the scurvy of animals, it is important that particular attention should be devoted to investigation of these organs. In a case of fatal infantile scurvy, focal degeneration of the lumbar cord has been described recently by Hess, the outstanding feature being a loss of cells in the lateral groups of the left anterior horn. The peripheral nerves are frequently the site of hemorrhage. This is well demonstrated in the recent work by Aschoff and Koch on the pathology of adult scurvy.

Experimental Scurvy.—In 1895 Theobald Smith described the development of scurvy in guinea pigs fed with cereals without the addition of grass or succulent vegetables. He did not, however, pursue the subject any further. In 1903 Bolle published the results of experiments on guinea pigs, an investigation which was repeated by Bartenstein. The results of these studies were not definite, as it was not clear whether true scurvy had been brought about, or merely a fragility of the bones, as the result of osteoporosis. In 1907 the first systematic and convincing study of experimental scurvy appeared. As the result of Eijkman's work on polyneuritis, a disease resembling beriberi, Holst and Froelich carried out a series of well-planned experiments on the pathogenesis of scurvy. This work was highly successful, and has been the basis of similar experimental investigations during the past decade. Most of these studies have been carried on in England and America, and have resulted in greatly increasing our knowledge of this disorder, more particularly our understanding of the antiscorbutic foodstuffs, of their relative potencies, and of the factors which tend to influence their activity. The guinea pig has been employed in most of these investigative studies. Scurvy has been produced in monkeys, but for some inexplicable reason it cannot be induced in rats, mice, rabbits, pigeons, or in fowl, although they receive what may be termed a typical scorbutic diet. The scurvy of the guinea pig is in almost all respects the counterpart of that in man. The lesions are almost identical, and all antiscorbutic foodstuffs which lead to a cure in the one species have the same effect on the other. The main difference seems to be that the guinea pig is far more sensitive to this disorder. The pathology of guinea pig scurvy is essentially that of human scurvy; hemorrhages and separations of the epiphyses or fractures of the long bones dominate the macroscopic picture. The hemorrhages, however, are found rarely in the gums. The microscopic changes are also the same as described in infantile scurvy; the bones show the typical Gerustmark and Truemmerfeld zones. Recently there have been histologic studies of the teeth, notably by Jackson and Moore, and by Zilva and Wells, which seem to prove that a lack of antiscorbutic vitamin leads to necrosis and pulpar fibrosis of the teeth, which in turn suggests that this vitamin plays a rôle in the dental caries of children.

The Relation of Scurvy to Rickets.—As is well known, scurvy is today regarded as one of the so-called deficiency diseases, a group which includes beriberi, a disease attributed to a lack of the water-soluble vitamin, and xerophthalmia, an eye condition brought about in rats by placing them on a diet free from the so-called fat-soluble vitamin. Various other nutritional disorders are frequently included in this category, among them rickets, by some thought to be dependent also on a deficiency of this vitamin. The relation between rickets and scurvy is a subject which was discussed by Glisson, and which has been the center of controversy since his time. Some accepted Barlow's dictum that scurvy is an absolutely distinct and separate entity, others have declared that the so-called scurvy was merely a form of rickets, whereas a third group considered it a combination of rickets and scurvy. Personally, I believe that the inter-relationship between these two disease is not very close. Infants may develop scurvy in spite of a liberal quota of cod-liver oil, which must be considered almost a specific for rickets, and on the other hand, large doses of this oil likewise fail to alleviate, to any degree, the scorbutic signs or symptoms. Furthermore, orange juice, the sovereign prophylactic remedy and curative for infantile scurvy, is of no value in the treatment of rickets. The same failures have been encountered in the treatment of scurvy in guinea pigs. This experience has been without exception, both from a prophylactic and curative standpoint. I am, therefore, of the opinion that these two disorders are absolutely distinct and that the confusion between them has resulted from the fact that the majority of infants in the temperate zone have rickets to some degree. Possibly a scorbutic infant may be slightly predisposed to rickets. This question cannot be determined definitely until an animal is found which is susceptible to both disorders.

Viewed from the pathologic standpoint, scurvy and rickets present strikingly dissimilar pictures. The former is characterized by almost complete cessation of activity of bone-forming elements, whereas the latter is distinguished by a hyperplastic condition leading to a marked overgrowth of cartilage and of abnormal bone.

On the Relation of Scurvy of Infants to that of Adults.—The identity of scurvy in the infant, in the young child, and in the adult is thoroughly established and requires no further substantiation. There are, however, certain differences between the symptoms of adult scurvy and those of Barlow's disease. These distinctions are due largely to the fact that the former disorder affects mature tissues, whereas the latter is engrafted on tissues which are in the process of rapid growth and development. The symptomatology is influenced also by the striking differences in environment—the passive, shielded existence of the infant contrasted with the active exposed life of the adult. The etiology in young and old is identical and the reaction to foodstuffs the same. It is remarkable that the question of the identity of adult scurvy and of Barlow's disease has been discussed for so long a time, and has been settled only within the last few years. In the German literature we still find considerable space devoted to a discussion of this aspect of the subject. The reason for questioning whether we may be dealing with two different disorders was the variable occurrence of hemorrhages of the gums, which are such a marked feature of the disorder in the adult, but are so insignificant in the scurvy of the infant. It is strange that so much emphasis has been laid on this minor divergence in symptomatology. For some time it has been evident that the involvement or non-involvement of the gums depends in the first instance upon whether teeth are present. In an infant having no teeth or none in the process of eruption, the gums will remain unaffected. Likewise in old people who are edentulous the gums do not become hemorrhagic or pouting. A second factor leading to a quite different intensification of this sign results from the fact that infection is usually present in the case of adults—that the gums are the seat of bacterial invasion.

Another sign which is more frequently encountered in infants than in adults is the separation of the epiphyses. We should expect this to be so in view of the great activity of the epiphyseal junction in infancy. This sign, however, is by no means uncommon in young adults, as noted by the celebrated surgeon Poupart in the last century. Subperiosteal hemorrhages are likewise more frequent among infants.

In adult scurvy we find more often large hemorrhages beneath the skin, in the muscles, especially the calves of the legs, and in the sheaths of the hamstring tendons and in the tendo achilles. These result naturally from the increased trauma to which adults are subjected. Moreover, syncopy, even of fatal character, has frequently been described in connection with adult scurvy, but is not mentioned in relation to infantile scurvy. These minor differences are merely such as we should expect to encounter in the course of a disorder occurring in individuals of such divergent ages and modes of life.

PATHOGENESIS AND ETIOLOGY

Pathogenesis.—There have been numerous theories regarding the etiology of scurvy—the lack of potassium, the lack of citric acid, acidosis, etc., theory. It is hardly necessary to consider each in detail, as experience has shown them to be untenable. About a generation ago, many children's specialists were of the opinion that the antiscorbutic potency of foods was due to their citric acid content, that cow's milk led to scurvy after having been heated because of its loss of citric acid. It was soon proved, however, that the loss of citric acid in the course of boiling was negligible from a therapeutic point of view. The acidosis theory was also short-lived, as it could be demonstrated that the addition of alkali to the food did not render it capable of preventing the onset of scurvy. The current theories of the pathogenesis of scurvy are three in number. There are many who still hold to the *toxic theory*. The minority report of the committee of the American Pediatric Society stated (1898) that "scurvy appears to be a chronic ptomaine poisoning due to the absorption of toxins." Neuman, one of the closest students of this disorder, considered infantile scurvy a chronic poisoning. It must be evident that the hypothetical poison is not introduced preformed in the food. In the first place, scurvy frequently develops among the babies of the well-to-do, who are receiving milk of excellent quality; it is not pre-eminently a disease of the poor, differing in this respect from rickets. Furthermore, there is no parallelism between the amount of the food consumed and its liability to induce scurvy. For example: We found that among infants fed pasteurized milk from a common source, if some receive diluted milk and others whole milk, the latter group will show less tendency to develop scurvy than the former, a result which would not follow were the poison contained in the food. Indeed it is by no means uncommon to encounter scurvy in an infant which has been receiving highly diluted milk. These considerations render it evident that the poison (if poison there be) is not introduced as such in the food. Some are of the opinion that the injurious toxins are formed in the intestine. In this connection it should be borne in mind that although many infants suffering from scurvy are constipated, this symptom is by no means constant or typical of scurvy. The disorder cannot be cured by a laxative, nor does it become worse on the administration of opium. Moreover, malt soup preparations, which in our experience have led to scurvy more frequently than any other food, are definitely laxative in character. Furthermore, orange juice, the great therapeutic mainstay in infantile scurvy, cannot be regarded as a laxative, and potato, another most valuable antiscorbutic, has no laxative properties whatsoever. The flora of the intestine, both in the experimental scurvy of guinea pigs and in the scurvy of infants, show no overgrowth of putrefactive bacteria and lend no basis to the hypothesis of ptomaine intoxication. A study of this subject by Torrey and Hess showed bacteria such as might be expected to be present on a diet rich in carbohydrates, and no significant changes after the scurvy was cured. The *bacterial theory* is still held by perhaps the majority of physicians in Russia, where adult scurvy is endemic, but where infantile scurvy seems to be uncommon. There is little basis for this theory except that the disorder frequently makes its appearance in epidemic form, resembling the sudden explosions of the infectious diseases. The error of interpreting all "epidemic" diseases as of infectious origin is well illustrated by the history of beriberi, which for years was held to be bacterial in origin. There have been but few blood cultures carried out in the course of either adult or infantile scurvy, but such as have been taken have almost always proved negative. In one of my cases as well as in one recently reported by Moore, the streptococcus viridans was cultivated from the blood stream during life. In cases of adult scurvy Czerny and Keller obtained no bacterial growth from blood aspirated from affected joints.

There can be no doubt that infection plays a rôle in scurvy, a very important rôle, in the complications which may lead to the death of the patient. The invasion of the blood, however, occurs after the disease is developed. It must be regarded, therefore, as an important secondary phenomenon, but one which is unessential from an etiologic standpoint. The relation of the chemical and bacterial factors furnishes an excellent example of the inter-relationship between nutritional and the infectious disorders, demonstrating that they are so closely interwoven that it is frequently impossible to distinguish between their clinical effects.

The newest theory, and the one held most widely at present, is the *vitamin theory*. It is an old theory dressed in new form—a scientific explanation for the well known fact that scurvy can be prevented and cured by means of fruits and vegetables, and comes about on a dietary which does not contain fresh food. This fact, well known not only to the physician, but to the layman as well, took more definite and scientific form after the experiments of Eijkman in 1897, who showed that polyneuritis in fowl was due to a food deficiency. The work which established this theory on a truly scientific basis in relation to scurvy was the classic investigations of Holst and Froelich. The numerous studies of the past decade have all served to strengthen this viewpoint, so that it may be stated without fear of controversy that the main pathogenetic factor in scurvy is a lack of the antiscorbutic vitamin. There are probably other contributory factors. As stated, bacteria may gain an entrance to the tissues through the intestinal wall, or perhaps toxins be absorbed after nutrition has been disturbed. It is also possible that the efficacy of the vitamin is in some degree affected by the composition of the food, by the corelation of its constituents. In my experience there has been no clear interrelationship between the amount of carbohydrate in the food and the development of scurvy, such as is believed to exist in regard to the pathogenesis of beriberi.

The Antiscorbutic Vitamin.—The recognition of the vitamins and of the accessory food factors is of very recent date. It has been realized, however, for many years that vegetables and fruits contained some factor which was not present in other foodstuffs. Such being the case, it is quite remarkable that it was not realized that this peculiar potency was associated with some heretofore unrecognized substance or factor. At the present time two other vitamins are well established—the so-called "water-soluble vitamin" to the lack of which beriberi is attributed, and the "fat-soluble vitamin," a deficiency of which leads to keratomalacia. The antiscorbutic factor is distinguished by being the most sensitive of these three vitamins, the most unstable and the least resistant to most physical and chemical processes. It is soluble in water and in alcohol. Vedder has recently shown that it is soluble also in acetone. He believes that it is an active reducing agent and has a strong affinity for oxygen. Holst and Froelich have shown that it passes through a dialyzing parchment and Harden and Zilva that it can pass through a porcelain filter. One of its most distinctive characteristics is its sensitiveness to even moderately high degrees of heat. In this respect it differs markedly from the water-soluble or so-called anti-neuritic vitamin, which withstands exposures to high temperatures. The reaction of the antiscorbutic vitamin in this regard is, however, not a simple one and cannot be expressed by a mere statement of the degree of heat and the length of exposure. Numerous other factors, especially the reaction of the medium, but also the physical environment, must be taken into consideration. For example: An acid medium greatly protects the antiscorbutic vitamin, as is shown by the resistance of orange juice or tomato juice. In relation to heat it has been found that the intensity of the heating process is not as destructive as its prolongation; for example: Boiling for a few minutes does not destroy the vitamin to as great an extent as heating to 60°C. for half an hour. Such results agree with the clinical experience that milk which has been boiled for a short period does not induce scurvy as readily as pasteurized milk.

Drying is generally destructive to this vitamin. However, it is incorrect to state, as does the British Medical Research Committee, that "it may be regarded as an axiom that dry or dried foodstuffs will not prevent scurvy." For dessication does not necessarily destroy the vitamin and some dried foodstuffs may still retain their antiscorbutic property even after drying.

There is one factor to which this vitamin is peculiarly sensitive, but which has been given no attention in connection with this subject. I refer to *oxidation* and oxidative processes to which attention has been called recently by Hess. A simple experiment will illustrate this fact:

> If a small amount of hydrogen peroxide, only as much as 4 c.c. of a normal solution, are added to a liter of raw milk, the antiscorbutic vitamin will be almost entirely destroyed after the milk has been kept in the incubator overnight. This can be readily demonstrated by feeding 80 c.c. per capita of this milk to guinea pigs and comparing the effect with parallel feeding experiments with similar milk to which no hydrogen peroxide has been added. In the former case scurvy will develop in three to four weeks; in the latter instance the animals will either remain well or not develop scurvy for some months.

This oxidation probably explains many discrepancies as regards the antiscorbutic content of food. It probably accounts for the fact that in some instances sterilized or pasteurized milk has led to the development of scurvy, whereas in other cases it has been borne without causing any untoward symptoms. For example: It is possible that the excellent results of Variot can be explained on this basis. It will be remembered that this French physician has frequently stated that he has fed thousands of infants on sterilized milk and never encountered a case of scurvy. Attention, however, has never been directed to the fact that the milk to which he had reference had been sterilized in hermetically sealed bottles. Again, it may well explain the fact that some pasteurized milk has led to scurvy, whereas pasteurized milk from another source has not induced any symptoms of this disorder. It may well account for sweetened condensed milk retaining a large part of its antiscorbutic content, for this preparation does not come in contact with the air in the course of its preparation. It seems probable that if the action of oxidative processes is borne in mind, the loss of the antiscorbutic vitamin may be prevented in the preparation of many foodstuffs where under present conditions this vitamin is entirely destroyed; this may well be true in relation to the dehydration of vegetables, which may suffer far less loss in this respect if the dehydrating is carried out in a vacuum or under conditions where the oxygen is replaced by some other gas.

In view of the well known action of catalysts in increasing oxidative processes, and the frequent traces of copper in milk, Hess and Unger carried out animal experiments to ascertain the effect of additions of small amounts of copper to milk. They found that heating milk in a copper vessel greatly increased the destruction of its antiscorbutic potency. Milk frequently becomes contaminated with copper in the course of pasteurization or condensation. It seems, therefore, possible that traces of copper may contribute to the destruction of this vitamine and account, in some degree, for the varying content of this factor in heated milk. This question has important bearing on the equipment and apparatus used in commercial pasteurization. It is also possible that the copper ingested with the food, especially where cereals are added to the milk, may exert a deleterious effect in this regard.

One of the most interesting questions in regard to the antiscorbutic vitamin concerns its *mode of action,* the manner in which it prevents or cures scurvy. This question cannot be considered in detail. At first it was suggested that it exerted an enzyme action, but this modus operandi is not accepted at the present time. Some believe that its action is direct; that it may serve as a source of nutriment to the tissues or exert an antitoxic effect on toxic products, or function as a catalyzer. Others attribute its potency to an indirect action, believing that it functionates through the agency of one or more of the endocrine glands. As is well known, this subject is in a state of great confusion, so that it cannot be discussed at the present time with profit. There is but one established fact which associates scurvy with the endocrine glands, namely, the enlargement of the adrenal glands, which has been found by several investigators to occur in the course of experimental scurvy.

Whatever the nature of the vitamin and its mode of action, it seems to act in a quantitative way, small amounts being insufficient to protect animals, a larger amount protecting some animals of a group, but failing to protect others, and a still larger amount conferring absolute protection to all.

Another interesting question which also awaits solution is the fate of the *vitamin in the body.* As is well known, the antiscorbutic vitamin cannot be elaborated by cells, but must be supplied by the food. In fact this is an important part in the definition of vitamins; whether, however, the antiscorbutic vitamin may undergo partial destruction in the intestinal canal, or whether the cells get the entire benefit of all the vitamin which is ingested, has never been ascertained. It is possible that some of it is destroyed in the alimentary tract and that this to some extent may be the explanation of the idiosyncrasy which characterizes the development of this disorder. Nor is it known whether the cells are able to store this vitamin to a considerable extent. Animal experiments which I have carried out on guinea pigs seem to indicate that, at least in this animal, this accessory food factor cannot be stored so as to protect the animal for a measurable length of time. Recently Van der Walle carried out some feeding experiments to test the excretion of antiscorbutic vitamin in the urine. He fed four guinea pigs each 15 c.c. of fresh urine daily and was unable to delay the onset of scurvy. This amount is so small that it does not warrant a conclusion that this vitamin is not excreted in the urine, although other considerations render this probable. It is therefore clear that we know very little more about the vitamins than that they exist or rather functionate as accessory food factors. Their physiological or pharmacological effect and the rôle which they play in metabolism is at present shrouded in darkness.

Etiology.—A consideration of infantile scurvy is concerned almost entirely with the artificially nourished baby. There are numerous reports scattered throughout the literature of cases of scurvy developing in *breast fed babies.* There is no doubt that this may happen, but it is such a rare occurrence that we can generally exclude the possibility of infantile scurvy in a baby which is being nursed. Under exceptional conditions, such as recently obtained in Vienna during the war, scurvy may develop more often in nursing babies. The investigation of the American Pediatric Society included 10 infants who had been breast fed exclusively, and there are perhaps a dozen others to be found in the literature. Without discussing these cases in detail it is to be noted that almost all of them are atypical; they have occurred in very young infants, frequently only a few months of age; the hemorrhages have involved the upper extremities quite as often as the lower extremities; there have been large subcutaneous effusions; in many instances the mother was suffering from some debilitating disease such as tuberculosis or syphilis. Infantile scurvy can occur in a nursing baby, especially where the milk is scanty, but especially where the mother is receiving very little antiscorbutic food in her dietary. It is probable, however, that many of the reported instances are merely bacterial infections occurring in poorly nourished infants. Here again attention should be called to the paradox that in Russia, where this disorder is endemic, there are practically no reports of infantile scurvy in nursing babies. Whatever the explanation, this exemption demonstrates that scurvy does not occur frequently in the breast fed baby.

The development of scurvy is intimately associated with the question of the antiscorbutic content of cow's milk, for it is the *artificially fed infant* which par excellence develops scurvy. In a consideration of this question we must at all times bear in mind the fact that the deciding factor is frequently whether a large or a small amount of milk has been consumed. In almost all reports of the etiology of scurvy a quantitative viewpoint is entirely disregarded, little or nothing being stated to indicate how much milk the infant consumed. Another factor of importance is the initial content of antiscorbutic vitamin of the milk, whether it was rich or poor in this vitamin; for as we know, its potency will depend upon the fodder of the cows.

But, quite apart from these considerations, infantile scurvy frequently comes about because the milk has been deprived of much of its antiscorbutic potency. This may be a result of pasteurization. *Pasteurized milk* always contains less antiscorbutic vitamin than the corresponding milk before it has been heated. In 1914 Hess and Fish reported mild cases of scurvy among infants who had received milk heated to a temperature of 160°F. for 30 minutes, a degree claimed by the National Committee of Milk Standards not to destroy its chemical constituents. Subsequent observations published by Hess in 1915 with milk heated to only 145°F. served to confirm this conclusion. In 1901 a large epidemic of scurvy occurred in Berlin as the result of the use of milk pasteurized bv a large dairy. However, we must not regard pasteurization as a definite and clearly defined process. It involves in some cases not only subjection to heat, but oxidative processes which are destructive of the vitamin. Therefore, some pasteurized milk will lead to scurvy, whereas milk pasteurized under different conditions may contain sufficient vitamin to protect against this disorder. It may be said in general that the more the milk is handled from the time it leaves the cow until it reaches the baby, the greater the danger of loss of its antiscorbutic properties. Thus homogenized milk, which was so popular in Switzerland and in France, proved to be a frequent cause of scurvy as well as the "lait maternisé" which led to the first appearance of infantile scurvy in France.

Contrary to what is commonly believed, boiling milk for a few minutes does not produce a food which must be considered as scorbutic. The statistics of Variot, who fed for many years thousands of children with sterilized milk and never encountered a case of scurvy, proved that sterilization does not necessarily lead to scurvy; but here again, as in the case of pasteurization, a great deal depends on the method. If the milk is sterilized, as Variot carried it out, in hermetically sealed bottles, oxidation cannot take place and destroy the vitamin. A large experience with dried milk has convinced me that milk may be dried without losing much of its antiscorbutic potency. I have cured cases of scurvy by means of milk which has been dried by the Just hot-roller process; in one instance, with milk of this kind which had been canned for a period of a year. It is advisable, however, to give an antiscorbutic to infants who are receiving dried milk, so that we may be certain that the baby is receiving an optimal amount of this factor, and secondly, as there are many methods for drying milk which may render the final product most inadequate in its content of this vitamin.

Condensed milk is stated as being a frequent cause of scurvy. Such has not been the case in my experience. Indeed, I have found sweetened condensed milk which was able to cure a case of scurvy. However, if the milk is initially poor in antiscorbutic vitamin, if it has been poorly preserved, and an insufficient amount given, it may well lead to infantile scurvy.

The greatest number of cases of scurvy came about from the use of the *proprietary foods*. Among the cases reported by the American Pediatric Society, about 60 per cent. had been given foods of this description. They furnish two important causative factors—a small and inadequate quota of milk and the deteriorating influence of heat. Generally not more than 16 to 20 oz. of milk are given for the day's feeding, and in the larger cities this milk frequently has been pasteurized.

There is an additional factor which comes into play in this connection, namely, the effect of alkalization. It is well known that the antiscorbutic vitamin is peculiarly sensitive to the faintest alkaline reaction, especially if it is at the same time subjected to heat. Proprietary foods with but few exceptions are alkalin, having been rendered so by the addition of potassium carbonate or bicarbonate. As a result of these various factors, we can well understand the large rôle which proprietary foods play in the etiology of infantile scurvy in the United States and England, where they are so commonly employed.

Age Incidence.—From a clinical standpoint scurvy may be said to occur in infants durng the second half year of their lives. There is general agreement on this point; it is borne out by the investigation of the American Pediatric Society and by the statistics of various observers. It is impossible, however, both from the etiologic and the therapeutic standpoint to distinguish clearly between the age when scurvy is commonly diagnosed and the earlier stage when it is manifested as a general nutritional disorder. We must remember that scurvy takes from six to nine months to reach its acute stage, and that there must be a prolonged period of nutritional failure preceding the diagnosis. If we are to consider the age incidence from the standpoint of latent or subacute cases, then the incidence of scurvy falls some months earlier than if we have merely the acute cases in mind.

Season and climate play but little rôle in the etiology. It has seemed to me that the scorbutic symptoms improve during the summer more than at other times of the year. The influence of season no doubt was in part dietetic in nature, depending on the fodder of the cows and the varying antiscorbutic content of the milk. Ultra violet radiations which are of specific value in the prevention and cure of experimental rickets, have, in my experience, been unable to delay the onset or ameliorate the symptoms of scurvy in guinea pigs.

Economic status must be considered in relation to the etiology of infantile scurvy. It is by no means infrequent to encounter marked cases of this disorder among the infants of the well-to-do, due to the extreme precautions in sterilizing milk or to the addition to the formulas of expensive proprietary foods or to the prevention of the child's obtaining a chance bit of fruit or vegetable. In its florid form it is not a disease of the poor, as is rickets.

Undoubtedly there is a *predisposition to scurvy,* as probably there is to every nutritional disorder. This has been remarked by many observers and was apparent some years ago when scurvy developed among a group of infants fed on pasteurized milk. Finkelstein cites an instance of a family where scurvy developed in two children in spite of precautions taken to avoid a repetition of this mishap. In many of the instances cited to prove the existence of this idiosyncrasy, no account has been taken of the daily intake of the milk, which renders the reports less convincing. Malnutrition by no means predisposes to scurvy; for instance, infants suffering from marasmus or atrophy are not prone to this disorder. From a dietetic standpoint it has been thought that possibly an excess of carbohydrate in the diet may lead to the development of scurvy. In my experience this influence has never been clearly evident either in the clinic or in the course of animal experiments.

As regards the *exciting factors* of infantile scurvy, only one is evident, and that is the influence of infection. Where latent scurvy exists infection will readily come about and precipitate the onset of manifest or acute scurvy. This is true of adults as well as of infants, and probably holds good for the other deficiency diseases.

SYMPTOMATOLOGY

Infantile scurvy must be considered as presenting three types of symptoms, which will be grouped as *acute, subacute and latent.*

The stereotyped picture, and the one which this designation suggests, is that of the acute type of the disease. I have described this elsewhere as follows: "We have to do generally with a poorly nourished, pale infant with a peculiarly alert and worried expression. As we approach its bed it whimpers or cries out in terror. Frequently its posture is characteristic, as it lies quietly on its back with one thigh everted and flexed on the abdomen. Examination shows that one or even both thighs are swollen and exquisitely tender, or that there is merely tenderness, the baby shrieking at the slightest pressure upon the lower end of the femur. If teeth are present, the adjacent gums are red, swollen and bleed readily. This is the syndrome which the medical student is taught to carry away to guide him in his everyday practice." This is a striking picture and one which impresses itself in the memory, but it is not the common form of the disorder, which should be diagnosed before this stage is reached.

Subacute Infantile Scurvy.—The commoner form, which we have termed "subacute infantile scurvy," comprises a large number of symptoms which are inconclusive individually, and frequently escape correct interpretation. The affected baby is usually in the second half of the first year of life, and does not gain in weight or gains but slightly for weeks. It may be fairly well nourished, but is pale, or sallow, with perhaps slight edema of the upper eyelids. The mother or nurse complains that the child is irritable and peevish, and that the appetite is poor or capricious. The gums show a lividity or slight peridental hemorrhage, which on subsequent examination may be no longer visible, and may have consisted merely of a rim of crimson edging the borders of the upper gum. Attention may be called to tenderness of the lower thighs, which in some instances is definite, in others so ill-defined and fleeting that it is impossible to convince oneself of its significance or even reality. There may be slight edema over the crests of the tibia, of a kind which does not pit on pressure. The knee-jerks are almost always markedly exaggerated. The urine is diminished in volume but is generally normal or contains a trace of albumen and red and white blood-cells. The pulse is frequently rapid, and becomes markedly rapid and irregular on the slightest excitement. The respirations are also rapid.

Happily, infantile scurvy now-a-days rarely progresses any further, because treatment is instituted and a quick recovery brought about. However, before the true nature of infantile scurvy was understood, the case progressed even to the point of death, and this happens occasionally at the present time. The gums become markedly hemorrhagic and even necrotic, leading to the foul odor of putrefaction. Hemorrhages of greater or less extent develop in various parts of the body. There are separations of the epiphyses of the long bones, accompanied by exquisite tenderness and swelling. The general condition of the infant becomes worse, the appetite fails, and the general nutrition is so greatly impaired that intercurrent infection comes about and leads to death, unless the condition is alleviated by giving large amounts of some antiscorbutic food.

Some have stressed the importance of roentgenograms in the diagnosis of scurvy, especially of "the white line of Fraenkel," which is seen at the epiphysis of the long bones, usually of the femora. Reliance cannot, however, be placed on this sign, as it is frequently absent when the diagnosis is otherwise clear.

As has been previously stated, scurvy takes about six months to develop. It is therefore evident that there must be, from a clinical standpoint, a pre-scorbutic stage leading to what may be called **latent scurvy.** This form of infantile scurvy necessarily must be the most common type. It is, however, frequently impossible to diagnose at this stage. Indeed, there is need for some means of diagnosing scurvy and other nutritional disorders before they have brought about such marked alterations that they are apparent to ordinary clinical observation. Latent scurvy is characterized by pallor, or rather a peculiar sallow or muddy complexion, by a lack of gain in weight, fretfulness, leading to frequent whining and crying, a loss or capriciousness of appetite. The knee-jerks may be increased, and the respirations and pulse rapid ("cardio-respiratory syndrome"). The symptoms are suggestive and do not enable an absolute diagnosis to be made. The diagnosis is established in part by an investigation of the diet of the infant—whether it was rich or poor in the antiscorbutic vitamin. Most of the instances of latent scurvy which I have seen have developed on pasteurized milk, which contains some vitamin, but an insufficient quantity. The diagnosis is furthermore established by giving an antiscorbutic food, for example, orange juice or tomato juice, and noting the prompt disappearance of the above symptoms.

As stated, hemorrhages dominate the picture of scurvy. The commonest manifestation are the hemorrhages of the gums. These do not appear unless the teeth are present or in the process of eruption. Very rarely small gingival hemorrhages have been noted where scurvy has not been present and the nutrition is normal. The gums may merely be bluish red or show a hemorrhagic rim above the teeth, or in severe cases may be deep red and pouting and even necrotic, similar to the lesion found in adults.

The other characteristic sign of scurvy due to hemorrhage is the *subperiosteal hemorrhage* (Fig. 144). This involves most frequently the lower end of the femur but is not infrequently encountered in relation to the tibia and may occur in bones of the upper extremity. The hemorrhage usually manifests itself by a sudden swelling at the lower end of the femur or femora. The swelling is very tender and may be barely perceptible or so large that it renders the leg twice its normal size. It persists long after the gums have become normal and becomes harder and less tender, and may develop a consistency of bone. In this stage such swellings have been diagnosed as new growths and have led to incision or even to amputation of the leg. The subperiosteal hemorrhages may be clearly seen by means of the Roentgen-ray. The shaft of the bone appears surmounted by an elongated blood-clot.

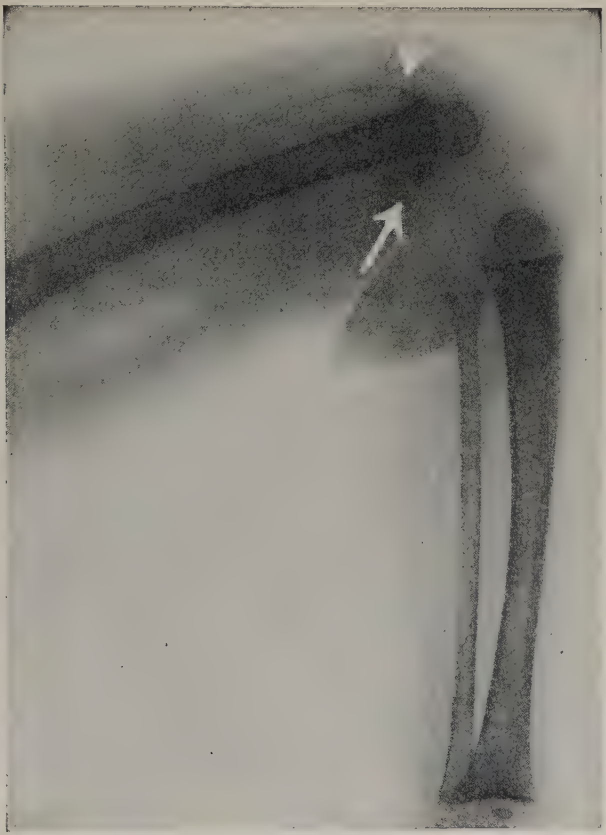

FIG. 144.—Subperiosteal hemorrhage at epiphysis of femur; beginning calcification of periosteum.

The periosteum frequently undergoes calcification, which renders its outline clear. Other forms or other manifestations of hemorrhage are the bleedings of the skin, which lead either to small petechial spots in various parts of the body, or to echymoses seen especially about the joints or on the forehead, following trauma. A form of hemorrhage which has always excited the interest of physicians, although it is uncommon, is the proptosis of the eyeball, which results from subperiosteal hemorrhage into the orbit. In general, it may be said that every organ has been described as being the site of hemorrhage—the pleura, pericardium, peritoneum, meninges; the glandular organs of the abdomen may at times show hemorrhages, depending largely on the occurrence of trauma or intercurrent infection.

A typical symptom of infantile scurvy is the separation of the epiphyses, which occurs most often in infants, but may be seen even in young adults (Fig. 145). It is most frequent at the lower end of the femur and upper end of the tibia. These epiphyseal hemorrhages are usually termed fractures or infractions. The union is remarkably perfect, even when no treatment has been employed and the cure has been effected by nature.

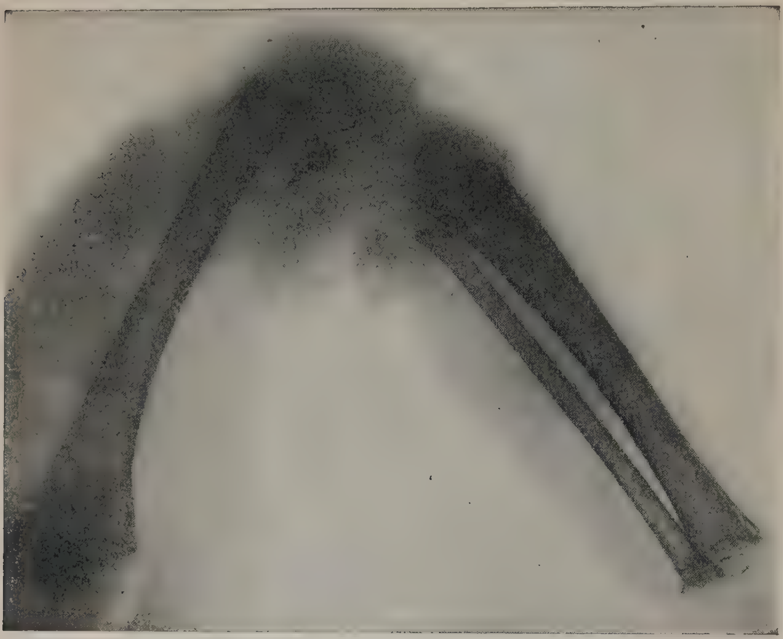

FIG. 145.—Separation of lower end of femur. Calcification of periosteum, appearing as "periosteal tags."

We have referred to the use of roentgenograms in connection with subperiosteal hemorrhages; it is also of value, as may be supposed, in substantiating the diagnosis of the separation of the epiphyses. The x-ray has been given particular prominence in this disorder in connection with the so-called *white line of Fraenkel.* By this is meant a transverse somewhat irregular band which is seen near the lower end of the epiphyses of the long bones, especially of the radius and the femur (Fig. 146). Its diagnostic value has been greatly exaggerated. We have found it frequently missing when the disorder is advanced and it persists for months after a cure has been effected.

Among the signs of infantile scurvy involving the bones *beading of the ribs* must be mentioned. Hess and Unger have recently described this sign, which has been considered as pathognomonic for rickets. It is important for us to realize that there is scorbutic as well as rachitic beading, and that the diagnosis of rickets cannot be based merely on the presence of a "rosary." The beading is somewhat different from that found in rickets, being more angular and less knobby.

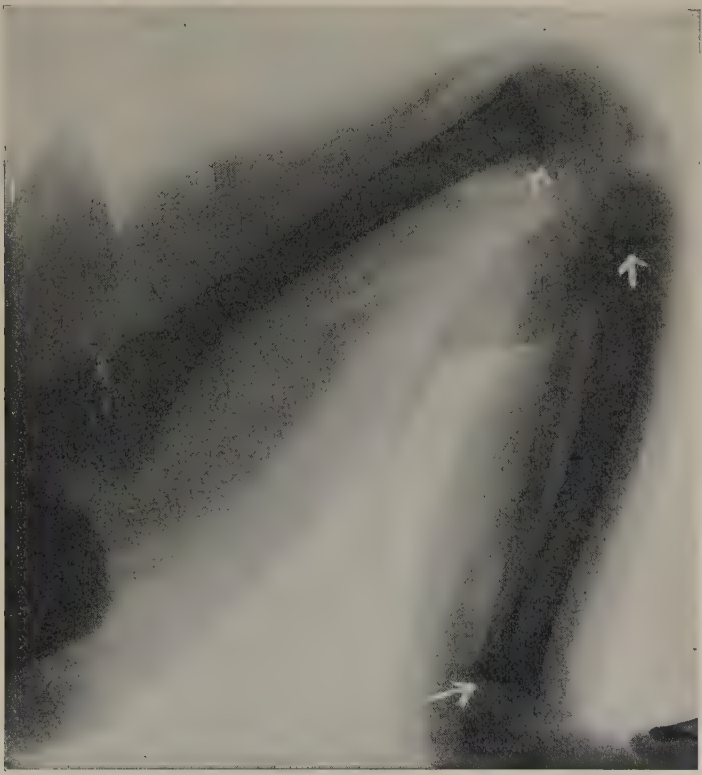

Fig. 146.—"White line" at epiphyses of femur and tibia.

The cardiovascular system is frequently involved in infantile scurvy. The heart is enlarged, as may be elicited by means of percussion and roentgenologic examination. The pulse is often rapid and unstable, increasing inordinately on the slightest exertion or rise of temperature. The respirations are also rapid. This symptom complex has been termed "the cardiorespiratory syndrome." The urinary system is frequently involved in the course of scurvy. Hematuria may occur as the first symptom. This fact is emphasized in many descriptions of the disease. It is an exceptionally rare sign, however, just as is hemorrhage into the orbit. Microscopic hemorrhage leading to the appearance of red blood-cells in the urine is, however, a frequent symptom, having been found by Still in 89 per cent. of his cases. Quite apart from or in combination with these hemorrhages there may be a true nephritis, accompanied by albumen and casts and irregular fever. This nephritis generally subsides quickly with the disappearance of other scorbutic symptoms. Oliguria is a symptom common to both adult and infant scurvy, but has been overlooked until attention was directed to it recently by Gerstenberger and by Hess and Unger. It gains an added interest in view of the fact that this symptom is common to both adult and infantile beriberi. When antiscorbutic food is given there may be a sudden outpouring of urine, leading to a loss of weight, which is quite unaccountable unless this explanation is borne in mind.

The symptoms of the gastro-intestinal tract are general rather than specific. One of the earliest manifestations of scurvy is a lack or capriciousness of appetite, which has been thought to depend on a decrease of the secretions of the gastro-intestinal tract. There is no regularity in the function of the bowels. In the report of the American Pediatric Society the bowels are stated as having been regular in 74 cases, irregular in 15, constipated in 126, and diarrheal in 65. There is no intimate relation between the constipation and the development of this disorder. Occasional hemorrhages occur from the bowel. Still more rarely hemorrhages may develop in the abdomen, especially under the peritoneum, and give rise to the diagnosis of peritonitis or appendicitis, leading to operative procedures.

Scurvy has been frequently classed as a hemorrhagic disease, and, as we have seen, hemorrhages play an important role in its symptomatology. This, however, is not due to a decrease in the coagulability of the blood. An investigation of this aspect carried out by Hess and Fish showed merely a slight delay in coagulation time. The bleeding time carried out by the simple method of Duke was slightly increased. The blood-vessels, however, do not possess their normal integrity and yield to increased pressure, as shown by applying a tourniquet about the upper arm (capillary resistance test). This may be due, as Aschoff and Koch suggested, to a lack of some colloidal substance needed for the normal structure of the vessels.

The blood itself usually shows secondary signs of anemia. In some cases the number of red cells is normal, or even excessive, in spite of the fact that the hemoglobin is reduced in amount. The leukocytes are normal where infection is absent.

Nutrition and growth are generally decidedly affected in infantile scurvy. The baby does not gain in weight and also does not grow normally in length. We do not meet with the apparently healthy infant, as we do in rickets. If the infant has been underfed for a long period an increase of weight may be brought about despite the fact that the food is deficient in antiscorbutic, and the scorbutic process is continuing. For many years there has been a difference of opinion whether there is a true scorbutic fever, or whether the mild fever which so frequently accompanies scurvy is a complicating process.

In view of the fact that intercurrent infections occur so readily in this disorder I think it is wise to consider febrile processes due to the action of bacteria and not to faulty nutrition.

DIAGNOSIS

A correct and early diagnosis of scurvy is important in view of the fact that we possess a specific remedy and that the disorder is not self-limited. Some years ago when infantile scurvy was little understood by physicians and cases occurred in greater numbers, the errors in diagnosis were very frequent. Even today the percentage of error is unusually high. If the subacute cases are included perhaps a majority of the cases are overlooked. It is remarkable how medical students, who have been taught the symptoms of this disorder almost invariably fail to recognize the first case which is shown to them in the clinic. It should be constantly borne in mind that where the diagnosis is uncertain the most important aid is an exact knowledge of the previous diet and observation of the reaction of the child to antiscorbutic treatment. The cardinal error is confusing infantile scurvy with rheumatism. There is little excuse for this mistake, as rheumatism hardly ever occurs at the age when infantile scurvy prevails, a case of rheumatism in a child of one year being a very great rarity. Nevertheless, Holt writes: "In fully four-fifths of the cases which have come to my own notice rheumatism has been the previous diagnosis." Such likewise has been my experience.

Another disease with which infantile scurvy is confused is purpura, owing to the fact that the gums may be hemorrhagic and swollen in both, and that there may be scattered subcutaneous hemorrhages and apparent pains in the limbs. Almost always a close inquiry into the previous diet will aid in establishing the diagnosis. It should be remembered that in true purpura the blood platelets are almost always reduced to less than 200,000, whereas in scurvy they are over 300,000 per cu. mm. At times both conditions may be present, as was experienced some years ago in what may be termed an epidemic of infantile scurvy. In this "epidemic" the scorbutic condition was latent and was complicated by an infection of so-called "grippe," leading to the occurrence of many subcutaneous hemorrhages in various parts of the body.

Cases occasionally are diagnosed as congenital syphilis, the pseudoparalysis being mistaken for the subperiosteal hemorrhage. It should be remembered that this syphilitic lesion occurs almost always before the fifth month; that it is preceded by a papular eruption and generally accompanied by a bilateral enlargement of the epitrochlear nodes and a positive Wassermann test.

A serious diagnostic error is confusing infantile scurvy with arthritis. There are many instances scattered throughout the literature where joints (especially the knee-joint) have been incised for supposed purulent arthritis and where a hemorrhagic condition was found. A still more serious error is confounding the swelling at the lower end of the femur with bone tumors, generally osteosarcoma. As mentioned above, the subperiosteal swelling in the course of healing may undergo calcification and develop bony hardness. If the previous course and history of the condition are unknown, a physician may mistake this swelling for a local malignant condition. It has happened that an amputation of the leg has been resorted to under this misconception. Surgeons should bear infantile scurvy in mind when called to perform operations for osteomyelitis. Other conditions which have been confused with infantile scurvy are acute endocarditis, neuritis, hemorrhagic nephritis, orbital tumor, appendicitis, peritonitis, acute poliomyelitis, Pott's disease, leukemia. Great caution should be exercised in diagnosing scurvy in breast fed infants. The great majority of reported cases are not scurvy, but bacterial infections, syphilis or various forms of intoxication. In establishing a diagnosis the same dietetic rules hold as in the case of bottle fed infants.

PROGNOSIS

The outcome of infantile scurvy is almost always favorable; accordingly it plays but an insignificant role in mortality statistics. This follows from the fact that we possess a specific remedy for its cure, so that if the diagnosis is made in time and treatment is prompt and thorough, the infant rapidly recovers, suffering no physical injury. However, where antiscorbutic foodstuffs are not procurable, as was the case in Vienna during the recent war and in other cities of the Central Empires, infantile scurvy occurs not only more frequently but is a not infrequent cause of death. In considering the mortality of scurvy and similar nutritional disorders, we must remember that they frequently predispose and lead to infection, and that therefore deaths which are attributed to such diseases as pneumonia or enteritis may fundamentally be due to scurvy, which furnished a fertile soil for the invading microörganisms. It is probably true that such was the case in the Central Empires during the war, for where there are many cases of frank scurvy there must be a far greater number of instances of latent scurvy predisposing to infection and leading to death. It thus becomes impossible to compute the mortality of scurvy. Still writes that in 5 out of 64 cases the disease proved fatal by diarrhea and exhaustion. The American Pediatric Society reports 29 deaths out of 397 cases, which gives a similar mortality percentage. When infantile scurvy was not so generally recognized by physicians the mortality was much higher, and we find that Barlow encountered seven deaths in his first series of 31 cases. As is well known, in adults the heart may be weakened by scurvy and sudden death may come about by cardiac failure. The same is true in infantile scurvy, although this occurrence is far more rare. Such involvement might well be expected in view of the tachycardia (the cardio-respiratory syndrome) which, as has been stated elsewhere, is so frequent a symptom of infantile scurvy. The heart may be rapid for months or even for years after the disorder, and tachycardia develop on the occasion of even a mild infectious disease. For example: A fever of 101 degrees may bring about a rise in the pulse to 180 beats. the usual course, however, is for the infant to respond almost miraculously to treatment. There are few reactions which are so prompt and satisfactory as the beneficent effects which come about on giving an antiscorbutic to an infant suffering from scurvy. Frequently within 48 hours or even less there is not only a disappearance of some of the symptoms, such as hemorrhages of the gums, but there is what is more important and striking, a complete change in the general condition and temperament of the infant. The nurse or mother notices that a smile has replaced the anxious look of the infant, that its appetite is much improved and that it is no longer sensitive to the touch. It should be borne in mind, however, that from a pathologic standpoint the cure is not simultaneous with the magic disappearance of the clinical symptoms, and that months probably elapse before the tissues return to their normal state. That such is the case is evident from radiographs, which not infrequently show changes in the long bones long after all other signs of scurvy have disappeared. The local signs usually disappear without leaving any trace. The hemorrhages are absorbed, the eczema disappears in a few days, the fractures unite promptly with the formation of a firm callus. The red blood-cells disappear from the urine, which usually gives no further evidence of renal damage, although in some cases a trace of albumen has been found in the urine some years after an attack of scurvy. It is difficult to give a definite opinion regarding the susceptibility to recurrences of scurvy in adults. Lind, who had the largest experience in this field, has expressed the opinion that one attack rendered a man more liable to a second attack. I have seen several cases where infants developed scurvy for the second time, but it was difficult to draw the conclusion that this was due to a heightened susceptibility. What makes me doubtful in this regard is an observation of several cases where the antiscorbutic food, contrary to the physician's orders, was omitted from the dietary, but where the infants did not develop scurvy for the second time; they did not thrive, but definite symptoms of scurvy did not reappear. It may well be that the question of a tendency to recurrence depends upon the individual idiosyncrasy to scurvy, which plays its rôle not only in the first but in subsequent attacks.

TREATMENT

The **prevention** of infantile scurvy, especially in its latent form, is of great practical importance, because of its frequent occurrence. There is no question but that human milk and raw cow's milk generally furnish sufficient antiscorbutic vitamin, unless the infant is greatly underfed, but there is a sharp difference of opinion as to whether pasteurized milk or milk that has been boiled or sterilized milk is adequate in this respect. As has been stated elsewhere, this divergence of opinion is largely attributable to the fact that clinicians have not considered the quantity of milk which they have given the infants, and also because they have regarded milk as a chemical entity from the vitamin standpoint, whereas, as we have shown, it differs greatly according to the fodder of the cows. Without entering into the details of this subject it may be stated that unless a baby is on a diet of raw cow's milk it should be given additional antiscorbutic foodstuffs. Furthermore, this supplement to the dietary should be carried out early so as not to allow the vitamin deficiency to exist for even a short period. My experience in this regard convinces me that there is no contra-indication to the giving of orange juice or of strained canned tomato to babies one month of age. At this time of life infants may be given one teaspoonful of orange juice or two teaspoonfuls of tomato, and this amount should be rapidly increased. Tomato is tolerated quite as well as orange juice, and has never led to any digestive disturbance.

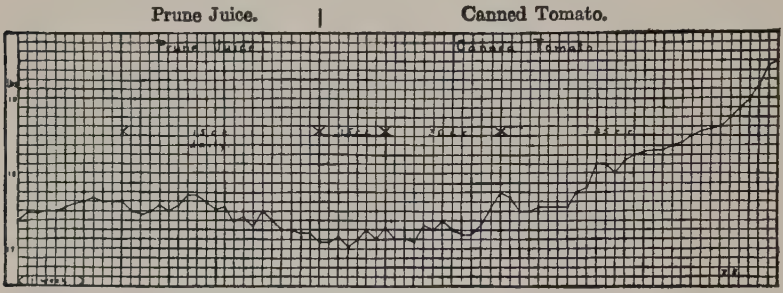

Fig. 147.—Cure of scurvy by the addition of canned tomato. In this case, as frequently as the alleviation of symptoms preceded the gain in weight. (From Hess, "Scurvy, Past and Present," J. B. Lippincott Co., Publishers.)

The common practice of physicians is to wait until the baby is about five or six months of age before giving an antiscorbutic, relying on the fact that it comes into the world with an adequate supply of this essential vitamin. This delay must, however, cause a gradual negative balance of this vitamin and allow a rudimentary scorbutic state to develop. Either of these antiscorbutic foods may be given, notwithstanding the fact that the baby has a tendency to loose bowels. Some infants who do not tolerate orange juice have been found to take the strained tomato juice without any disturbance. The amount of antiscorbutic should be increased so that when the baby is three months of age it receives one tablespoonful of orange juice or twice the amount of tomato. The tomatoes are merely strained through a collander and warmed (not cooked); the fluid portion is given and not the solid material, and may be administered by spoon or through the bottle (Fig. 147).

Another antiscorbutic which has been recommended for routine use to prevent infantile scurvy is swede juice prepared by grating the raw vegetable and squeezing the pulp in muslin. This juice has been adopted in some of the English welfare stations. It should be given in about the same doses as tomatoes, and from reports seems applicable in countries where the swede can be readily obtained. For babies over six months of age reliance may be placed on the ordinary vegetables such as potatoes, spinach, carrots, etc. Here again attention must be paid to the amount which is taken, and it should be realized that one or two teaspoonfuls of these vegetables is not sufficient to furnish an adequate quota of antiscorbutic vitamin. This is especially true if the vegetables are old or not fresh, or are cooked for a long period. Instances where scurvy developed in spite of the administration of vegetables are probably explainable by the fact that the daily ration was too small or that the vegetables were low in antiscorbutic potency. In giving baked potato it is commonly advised to use the floury part which is beneath the peel; this should be put through a sieve and thoroughly mixed with milk so as to form a cream. For younger babies it can be prepared with water to form a diluent similar to the barley water commonly employed to dilute cow's milk (a tablespoonful of potato to a pint of milk cooked for 15 minutes).

Rhubarb has been shown to possess effective antiscorbutic property, and as might be expected from its acid reaction, retains this potency in spite of boiling. This holds true for the fruit itself as well as for its juice. The same is true of strawberries. Ten cubic centimeters of strawberry juice was able to cure guinea pigs of scurvy even after it had been boiled. Banana is weak in its content of antiscorbutic vitamine; however, if a comparatively large amount is added to the dietary, it is possible to confer protection.

Little reliance should be placed on beef juice. Cases of scurvy have frequently arisen from the fact that physicians or mothers have regarded beef juice as an antiscorbutic. This same error has taken place in respect to cod-liver oil, due to a confusion of rickets and scurvy.

Curative Therapy.—There are few reactions more striking in the field of therapy than the prompt reaction to antiscorbutic treatment. A magic result is noted frequently within 48 hours or less. The baby which has had a poor appetite, has been irritable and tender to the touch, suddenly regains its appetite, is playful and can be handled without occasioning pain. Generally within a week, unless the case is severe, all symptoms of scurvy may have disappeared. The gums are no longer hemorrhagic and the infant appears normal and cured as far as can be ascertained by clinical examination. The table appended is reproduced from the report of the American Pediatric Society and shows the duration of treatment before marked improvement was noted (Table 1). In the cure of scurvy the same foodstuffs are employed as in its prevention. Orange juice is the sovereign remedy and should be given in a dosage of about 2 oz. a day. Canned tomato is also most efficacious and is sometimes tolerated when orange juice is not well borne (Fig. 147). If these additions are made to the dietary it is not absolutely necessary to alter the food if it is adequate in caloric value and is not deficient in other respects. In general it may be stated that in addition to giving fruit or vegetable juices it is well to give milk which is either raw or has not been heated to too high a degree or for too long a period. Antiscorbutic treatment should be continued for months as the clinical improvement far outstrips the restoration of the bones and other tissues. Orange juice may be given intravenously. This method may be resorted to in exceptional cases where food cannot be tolerated by mouth and in cases where there is extreme prostration. For this purpose the orange juice is obtained in as sterile a manner as possible, boiled for five minutes and is rendered neutral or slightly alkalin by the addition of normal sodium hydroxide just previous to its injection. Hess and Unger report three cases where this procedure was carried out without the slightest untoward reaction, and with very prompt subsidence of the symptoms.

TABLE 1.—DURATION OF TREATMENT BEFORE MARKED IMPROVEMENT

Days	Cases	Weeks	Cases	Months	Cases
1	19	1	47	1	6
2	58	2	27	2	4
3	46	3	8	3	4
4	26	4	1		
5	19	5	1		
6	1	6	1		
7	2				
8	2				
9	1				
10	7				
12	2				

Non-dietetic Therapy.—There is little to be done for the patient in addition to the giving of sufficient antiscorbutic. No drug has been found to have any antiscorbutic value; cod-liver oil, yeast, and the various extracts of the endocrine glands are without the slightest value. If the gums are markedly hemorrhagic and fungous they may be treated with nitrate of silver stick or tincture of myrrh. Fractures of the long bones should be supported by means of splints and given the usual surgical treatment. Under no circumstances should hemorrhagic infusions in or about the joints be incised. The baby should be placed in a room which is well aired and lighted and handled as little as possible. It is of the greatest importance that intercurrent infections should be avoided by shielding an infant from contact with those who may be suffering from infectious diseases, especially any respiratory infection.

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CHAPTER XXXI

PELLAGRA

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INTRODUCTION

How long pellagra has existed in the world is not known, although the date of its origin has been diligently searched for, but not always without prejudice.

It devolved upon those who held to the corn theory with reference to its etiology to prove that pellagra did not exist in Europe until after this article of diet was introduced into that country.

The zeists have, therefore, made much of the fact that in Gaspar Casal's "Natural History of the Asturias," written in 1735 but not published until 1762, is to be found the first description of the disease sufficiently accurate to permit its identification. Corn was introduced into Europe in the latter part of the seventeenth century.

Casal in this work described eight cases of "a very singular kind of leprosy," which he had observed in the Spanish province of Oviedo. To this disease, Casal applied the name *Mal de la Rosa.* According to Wood,[1] nowhere in his work does Casal claim or imply that he had coined the name, the inference being that the name was already in use by the people before Casal used it in his work. The rule governing the history of a disease is that it exists some years among a people before it is known to that people by any particular name and that the disease exists for many years before appearing in the literature of that people.

The acceptance by many present-day writers of the idea that pellagra is a comparatively modern disease is in a large measure due to the influence of Gaetno Strambio. The latter was in charge of the first hospital in existence for the treatment of pellagra. The hospital established by Joseph II of Austria in 1784 was operated for three years and abandoned. A greater hospital was opened in Milan and to it Strambio was called. It was in the Milan hospital that Strambio made most of his observations on the disease, observations which, because of accuracy in description, retain for their author the high regard of all subsequent investigators.

Strambio searched the writings of the ancients without success for a description of a disease sufficiently complete to fix it upon pellagra. Notwithstanding the fact that he was thereby convinced that pellagra was a modern disease, Strambio admitted that the older writings might contain disassociated descriptions, which, if possible to connect, would furnish a complete picture.

Frapolli, who was the first to use the name pellagra in literature, considered the disease an ancient one. He stated in his treatise (1771) that the name pellagra was the one he found applied to the disease by the Italian peasantry. (The derivation of the word is probably *pelle*, skin, *agro*, rough.) Frapolli believed he was able to discover evidence of the existence of pellagra as far back as the year 1573, at which time it was known under the name, pellarella. Although Strambio claimed the only similarity was in the name, no convincing evidence has been offered to disprove Frapolli's conclusion that these diseases were identical.

Important though it may be to fix upon the date approximating the first appearance of pellagra in Europe, other than the briefest summary of the discussion on this subject would be out of place in a text of this kind.

The majority of pellagrologists hold to the opinion that the disease is a modern one, making its first appearance in Southern Europe in the early part of the eighteenth century. They point with telling effect to the fact that the literature preceding this period contains no descriptive reference to pellagra; that all within a few years closely following appeared many descriptions of the disease: By Casal in 1735; Thiery in 1755; Frapolli in 1771; Odoardi in 1776; Pujati in 1776; Gherardini in 1780; Albera in 1780; Strambio in 1784; Townsend in 1787; and many others.

A small but equally brilliant number of investigators contend that pellagra confused and intermixed with leprosy, scurvy and syphilis was described by the writers of antiquity. The men holding this opinion have been able to bring out a similarity between the modern pictures of pellagra and certain symptoms of various disorders described by the ancients striking enough to keep the unprejudiced mind unconvinced as to whether the evidence on the other side possesses sufficient weight to swing the scales. Wood,[1] for example, calls attention to Hippocrates's description of *solicitudo*, in which the spring recurrence of slight delirium and melancholia bring it very close to our modern idea of pellagra.

The date of the appearance of pellagra in the United States has also never been definitely fixed. Some writers have held that there is evidence of its existence among the Indians at the time of the coming of the white man to the country.

In his presidential address to the National Association for the Study of Pellagra, Columbia, S. C., 1912, J. W. Babcock reported a case from the records of the South Carolina Insane Asylum as follows: "The patient was admitted, February, 1834, suffering from a second attack of insanity. He was violent, homicidal and suicidal. Soon after admission he developed insomnia and severe diarrhea. Ptyalism was noted. He was emaciated and had acute exacerbations of diarrhea, especially at night. In August the skin on dorsa of his feet became of somber brown color, suggesting purpura. In September the purpuric rash involved his hands and scurvy was suspected. In

October the diarrhea, which persisted in spite of treatment, is recorded as incorrigible at night. Later he became edematous, and purpura reappeared. In spite of antiscorbutic dietary, his diarrhea persisted, he became exhausted and expired on Nov. 22, 1834."[2] Babcock's citation of this case is given in full, for by it, we believe, he has adduced sufficient evidence to substantiate his claim that pellagra existed in this country earlier than is generally known.

A case of pellagra was reported by Dr. John P. Gray, Utica, N. Y., in 1864. In the same year another case was reported by Doctor Tyler of Somersville, Mass. Both cases were adults and insane.

Evidence is accumulating that the frightful morbidity and mortality among the Union prisoners in the Confederate prison at Andersonville, Ga., was to a considerable extent due to pellagra. Referring to his interviews, years after the war, with two former guards and a former prisoner, all men of intelligence, Niles[3] says: "(They) have told the writer how the men had a supposed eczema; how they loathed their food; . . . how their skins were rough and hard, and how their hands were sore and cracked; how their bowels were chronically loose, so much so that there was a pathetic joke that a prize would be given any prisoner having a solid fecal movement. . . ."

The nature of the disease was recognized and reported in a pellagrous Italian sailor by Sherwell of Brooklyn, 1883. In 1889, Bemis left a written diagnosis in the case of a white woman, a patient in the Charity Hospital, New Orleans. In 1902 Sherwell reported another case. In the same year, Harris, Atlanta, Ga., reported "A Case of Hookworm Disease, Presenting the Symptoms of Pellagra." In 1907, George H. Searcy reported an outbreak in the Mount Vernon, Ala., asylum for insane negroes. These cases, 88 in number, were studied by Searcy and E. L. McCafferty of the hospital staff during the years 1906 and 1907. Before reporting these cases, Searcy invited Eugene Bondurant of Mobile, and Isadore Dyer of New Orleans to examine them. This they did and they confirmed his findings. The cases were reported as pellagra at a meeting of the State Medical Association of Alabama, April 19, 1907. In the same year, but working independently, J. W. Babcock reported the existence of pellagra among the inmates of the State Insane Asylum at Columbia, S. C. In 1907 also, J. T. Searcy discovered its existence among the patients at the Bryce Hospital at Tuscaloosa, Ala.

In 1908, C. H. Lavinder of the United States Public Health and Marine Hospital Service, stationed at Wilmington, N. C., had his attention called to the disease by local physicians. Lavinder in turn reported the existence of pellagra to Surgeon-General Wyman. The latter ordered Lavinder to prepare at once a précis for publication. In 1906, the South Carolina Board of Health held a conference on pellagra. In 1909, chiefly due to the efforts of J. W. Babcock, the First National Pellagra Conference was held in Columbia, S. C.

Through these various agencies, a knowledge of the nature of the disease was quickly disseminated. As a result pellagra was recognized and reported in many localities of the different Southern States. At every contemporary medical meeting held in the South, pellagra became the live subject for discussion. Diagnoses on many cases previously regarded as psoriasis, eczema, syphilis, symmetrical gangrene, dermatitis, hookworm, dysentery, etc., were revised. Some of the older physicians in Alabama have told the writer that since becoming acquainted with the nature of pellagra, they could recall cases of the disease occurring in their practice many years back—some as early as 1865. It is very doubtful, however, if pellagra was ever prevalent in the United States prior to the year 1900.

While pellagra occurs sporadically in nearly every state of the union, the most intense foci are found in the South Atlantic States—Virginia, North Carolina, South Carolina, Georgia, Florida, Kentucky, Tennessee, Alabama, Arkansas, Mississippi, Louisiana and Texas.

Wide variations exist in the published estimates as to the number of pellagrins in this country. Deadrick and Thompson[4] estimated in August, 1915, that there were 25,000 cases of the disease in the United States. Niles[3] in January, 1916, placed his estimate at 40,000 cases. Lehmann[5] in November, 1915, said there were between 40,000 and 45,000 cases of pellagra in Texas alone. Such wide differences in the opinion of investigators can only be accounted for on the ground that few of the states in which pellagra is most prevalent furnish full and accurate vital statistics.

The latter fact, too, renders it impossible to definitely determine whether the disease is on the increase or the decrease.

ETIOLOGY

General Characteristics.—Some of the important facts relative to the nature of pellagra are as follows:

1. More females than males contract the disease. The rate of incidence among children under 10 years, however, is practically the same in both sexes.

2. There are probably more pellagrins among the whites than among the blacks. This fact is believed to be due to conditions other than any racial resistance enjoyed by the negro.

Wolff[7] has called attention to the fact that Jews are practically immune, the disease occurring in that race with exceeding rarity.

3. The disease is much influenced by season. Appearing in the spring and early summer, it reaches its period of maximum activity in midsummer, after which it begins to decrease and may almost disappear during the winter months. Symptoms in pellagrins are much relieved and often disappear in cold weather, only to reappear in the spring or summer following. Recurrence is estimated to take place in 60 per cent. of cases. The Thompson-McFadden Pellagra Commission[14a] discovered no particularly marked tendency for the seasonal recurrence to appear during the same month year after year. Relapses were observed in the fall months of the same year in which the onset appeared.

4. It has been discovered that pellagra, in the United States at least, depends neither on altitude nor on topography. The disease exists in mountains and in lowlands. It is found in arid regions and in swampy districts. It is met with in proximity to creeks and, as often, far away from creeks.

5. Pellagra is not contagious. Officers in institutions, among the inmates of which pellagra may be rampant, do not suffer. Doctors, nurses, and other attendants, brought into intimate contact with pellagrous patients, do not contract the disease.

6. Pellagra as a rule is found amidst insanitary and unhygienic surroundings. Such surroundings, however, are not essential. Pellagra at times attacks individuals who live under modern hygienic conditions. But no hot bed of the disease has ever been discovered except associated with poverty, not necessarily abject, but where the necessities of life alone are had. Excepting possibly a few institutional outbreaks, no hot bed of the disease has been discovered elsewhere than in unhygienic surroundings.

7. Pellagra occurs more frequently in the rural population than in the population of cities. Roberts, however, was too dogmatic in his statement, "Pellagra stops at the city gates." A number of competent observers have, for some time now, been reporting cases occurring in city dwellers. Pellagrous outbreaks, moreover, are more, frequent in penitentiaries, insane asylums, orphan homes mining camps, cotton mill and factory villages than in the country proper.

8. Malaria, hookworm infection, amebiasis, etc., are conditions frequently concomitant with pellagra. Ofttimes pellagra develops after prolonged lactation, tuberculosis, typhoid fever, venereal disease, or gestation. Any ailment causing a rundown condition seems to predispose to pellagra. Alcoholics are apparently more susceptible than are persons of temperate habits. Strambio mentioned the fact that rachitic children and chlorotic girls were particularly susceptible. H. W. Rice,[8] discussing pellagra as it existed in two orphanages, brought out the interesting fact that in both institutions an epidemic of measles and of whooping-cough occurred just prior to the outbreak of pellagra.

The Thompson Pellagra Commission in a recent communication[16g] states that, although there is an increased liability to the development of an initial attack of pellagra in the earlier months following childbirth, pregnancy confers on the pellagrous woman an increased resistance to recurrence.

In an earlier report,[14b] the Commission announced that the development of pellagrous symptoms in a number of children was immediately preceded by one of the acute exanthematous diseases.

9. Pellagra is not inherited. In his many observations in Italy and elsewhere, Sambon[9] never saw a case of congenital pellagra. Moreover he was unable to find any authentic report of such a case in the literature.

On the other hand, the majority of investigators believe that pellagra in the parent predisposes the child. Roberts graphically pictures the effects of a pellagrous parentage on its offspring as follows: "The pellagrin suffers not only a chronic neurasthenia, an ever increasing tendency to melancholia, but also actual organic changes in the cord and brain. These organic changes do not appear in the child but the stigmata of degeneracy do appear. Pellagra is not inherited, but the result of its ravages in the parent is inherited, and appears in the child in the form of deficient development, anemia, various malformations of the skull, asymmetry, bad-set ears, mental weakness, slow growth, an unusual lack of resistance and a frailness out of all proportion to age. In addition to pellagra, the parent may also have ankylostomiasis, or be tubercular, syphilitic or alcoholic, and the degeneracy in the child would thus be increased. Let this continue for two or three generations, and it is natural to find the descendants of pellagrins suffering with the widely prevalent disease of the community, and increasingly degenerate and pauperized. Pellagra thus becomes a real cause of race degeneracy. These children live under the very same conditions in which their parents developed pellagra, and this very degeneracy is, in turn, an invitation to the disease already widely prevalent. If strong men develop pellagra, frail children will develop pellagra much more easily. The injurious influences are at work, and both parents and children may be attacked at the same time, or children may become pellagrous first and their parents afterward."[10]

From this Roberts concludes that adult pellagrins should not marry. To this, as a general conclusion only and in so far as it pertains to that large collective class of poor folk in the South among whom pellagra is so rife, does the author subscribe. To the latter are known several formerly pellagrous adults, who are now absolutely cured, being as stable, sane, and sound as any other normal individual. He feels sure that there are many such persons throughout the country to whom to deny the happiness of marriage and of begetting children would be a senseless and a useless hardship.

The question of heredity was studied, for the Thompson-McFadden Pellagra Commission, by Dr. C. B. Davenport and Dr. Elizabeth Muncey. Davenport[16b] is inclined to regard inherited susceptibility as being pronounced. He holds the opinion that the progress and behavior of the infecting organism, granting pellagra is communicable, in the body of affected individuals depend, in part, on certain constitutional, inheritable traits.

Muncey[16c] thinks the inherited predisposition to pellagra is pronounced only in families in which chronic gastro-intestinal symptoms have existed for several generations.

10. No age is exempt from pellagra.

Dr. T. C. Savage[11] has reported an undoubted case of the disease in a two months old infant. Marcus Haase[12] has reported cases in infants four weeks, six weeks and eight weeks respectively. William

Krauss[13] has reported two cases in nursing infants. J. A. Hayne[13] has reported having seen the disease in a nursing infant. Sambon held to the opinion that pellagra in Italy was much more common in children than was generally believed. He states: "The youngest case I can mention is that of an infant, Luigi Anselmi, who developed the disease in 1911 when only three months old. Besides the three months old case, I have seen two other infants, who were attacked at the ages of four and five months, respectively. Such cases are somewhat rare, but the disease is quite frequent in children from six to eighteen months old."[9]

Up to 1912, Wood[1] had not seen the disease in an infant younger than 22 months. Niles[3] as late as 1916 had not personally encountered pellagra in children under five years of age. Deaderick and Thompson,[4] as late as August, 1915, had not seen pellagra in a child younger than two years of age. Roberts[10] does not believe that pellagra in the United States is as common among infants as it is in Italy, but he admits that closer study may reveal cases previously overlooked or not reported. The Thompson-McFadden Pellagra Commission summarizes the age incidence as follows: "Children under the age of two, adolescents for about five years following puberty, and adult males in the active period of life were least frequently affected by pellagra. On the other hand, women from 20 to 44 years of age, old persons of both sexes, and the children from 2 to 10 years of age were most frequently affected."[15a]

The youngest case of the author's in which there existed definite and recognizable manifestations of pellagra was in a child 26 months old. Several cases in young children, three to five years of age, have been seen, but the greater number of his cases have been in children who have passed the fifth year.

He has observed in a number of infants a condition somewhat similar to *pellagra universalis.* All such infants were found to be either nursing dry breasts or breasts which secreted a milk exceedingly low in both fats and proteins. This condition, to which the author has applied the term pellagroid, will be discussed more fully in another part of the chapter.

Unless it shall be discovered that the condition referred to in the paragraph above is to be classed with true pellagra, the author agrees in the opinion that the disease occurs with but extreme rarity in children less than five years of age, but that after this age it occurs with great frequency.

Theories of Etiology.—The cause of pellagra has to this day proven elusive. Since the time of Casal until the present, the cause of the disease has been an attractive subject for theorizing. Of the theories advanced, some are to be characterized as being nothing more sincere than the efforts of speculative men taking the gambler's chance on hitting it right; others, as the result of ingenuous but prejudiced reasoning; while some few are based on real painstaking and true scientific investigations.

Pellagra at one time or another has been thought to be due to:

1. Corn. Principal zeist theories. (Taken from Deaderick and Thompson.)
    1. Lack of nutritive elements.
    2. Toxicity of normal corn itself.
    3. Toxicity of corn due to germination process.
    4. Toxicity due to products elaborated by action of certain microörganisms.
    5. Toxicity of corn due to products produced in the alimentary tract after ingestion.
    6. Toxicity of corn due to poisonous substances in the grain which act only after being sensitized by rays of sun.
    7. Action of certain parasites of corn ingested with that grain.
    8. *Toxicity due to seed of noxious weeds gathered with the grain.
2. Lack of salt in the food. (Odoardi.)[1]
3. Poverty and bad hygiene. (Strambio.)[1]
4. Bad air; moisture, etc. (Thouvenel.)[1]
5. Actions of sun's rays. (Frapolli.)[1]
6. Lack of wine with food. (Gherardini.)[1]
7. Accumulation of heat in the blood. (Del Camp.)[1]
8. Accumulation of electricity in the blood. (Vay.)[1]
9. Accumulation of acids due to faulty excretion. (Boerhaeve.)[1]
10. Action of poisonous weeds on exposed surfaces of body (probably an old conception but one which the author finds still cherished by a certain class of people).
11. Semi-dried edible oils. (Mizell.)[3]
12. Sand-fly. (Sambon.)[3]
13. Buffalo-gnat. (Garman.)[17]
14. Stable-fly. (Jennings and King.)[14c]
15. Water-borne nematode. (Alessendrini.)[4]
16. Ameba. (Long.)[4]
17. Mosquito. (Roberts.)[18]
18. Pathogenic trypanosomes transmitted by blackbirds. (Ravitch.)[17]
19. Bacillus Welchii. (Holmes.)[19]
20. Mineral Acidosis. (Alessandrini and Scala.)[3]

The above theories are not given in chronological order nor do they embrace all that have been advanced. The enumeration, however, is sufficiently complete to suggest to the reader the reason for leaving out of this work further consideration of many of these theories.

It may be said that in a general way students of the etiology of pellagra in the United States are at present divided between two schools of thought.

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* No. 8 is not contained in Deaderick and Thompson's table but has been added by the writer.

The one school, represented by the Thompson-McFadden Pellagra Commission, leans strongly to the opinion that pellagra is a low-grade infection, primary lesions of which are probably located in the intestinal canal. It grants the great influence of dietary deficiency as a predisposing factor but contends that this factor in itself cannot satisfactorily explain pellagra.

The other school, headed by Surgeon Joseph Goldberger of the United States Public Health Service, insists that pellagra is in no sense a communicable disease and that the disease is solely due to some dietary fault.

The Thompson-McFadden Pellagra Commission, the conception of Dr. Geo. N. Miller, at the time president of the New York Post-Graduate Medical School, was organized for the purpose of sending out a research expedition for the investigation of pellagra as it existed in the United States. Funds for the conduct of the investigation in 1912 and 1913 were donated by Col. Robert M. Thompson of New York and by Mr. J. H. McFadden of Philadelphia. For the work done in 1914 and 1915 funds were provided by Col. Robert M. Thompson and Dr. Geo. N. Miller of New York. The Commission was constituted as follows: Captain J. F. Siler, Medical Corps U. S. Army; Passed Assistant Surgeon P. E. Garrison, U. S. Navy; and Dr. W. J. McNeal, Professor Bacteriology and Pathology in the New York Post-Graduate Medical School. The Bureau of Entomology of the U. S. Department of Agriculture detailed Messrs. A. H. Jennings and W. V. King to aid the Commission by investigating the possible etiologic relation between insects and pellagra. On the invitation of the Commission, a number of other investigators made studies on special features of the disease.

The Commission established field headquarters in 1912 at Spartanburg, S. C., and instituted an intensive study of the epidemiology of pellagra as it existed in Spartanburg County.

In addition to the field work, laboratory work was done in the New York Post-Graduate Hospital on material furnished by pellagrins sent there from South Carolina. The laboratory investigations were carried on by Drs. O. S. Hillman, R. M. Taylor, V. C. Myers, and M. S. Fine, under the supervision of Drs. Jonathan Wright and W. J. McNeal.

The purpose of the Commission was contained in this announcement: "The problem immediately before the Commission in all its work was the etiology of pellagra and not its symptomatology or therapeutics, except as these subjects might throw light upon the essential nature of the disease."[14]

The mass of data collected by the Commission is most valuable and interesting, but too voluminous to be summarized except in part in this volume.

Besides those already referred to in a previous part of this chapter, some of the important findings and conclusions of the Commission are as follows:

I. The not infrequent presence of a leukocytosis in pellagrins; suggesting the possibility of an infectious etiology of obscure origin or one due to complicatory disturbances. No characteristic or constant variations in the large mononuclear leukocytes and eosinophiles were noted. A lymphocytosis was observed in a majority of cases.[14f] Further observations on the blood count led to the later announcement that although a slight rise in the leukocytes was found in some cases during the first stages of an acute attack, a pronounced leukocytosis was observed in no instance. No constant abnormality was disclosed.[15e]

II. Excessive amounts of indol and especially skatol in the feces of pellagrins. The presence of excessive amounts of indican in the urine associated with high elimination of ethereal sulphates. These findings when taken together were regarded as indicating some unusual bacterial conditions with putrefaction high up in the intestines.[14e]

III. Many unusual bacterial strains were isolated from the pellagrous intestine. One (No. 67) of these strains when tested against the sera of pellagrins gave complete agglutinations in a large percentage of cases. However, positive results in a small percentage of cases followed the testing of this strain against the sera of apparently normal individuals.[14g, 15a]

IV. Ticks, lice, bed-bugs, cock-roaches, horse-flies, fleas, mosquitoes, buffalo-gnats and house-flies were eliminated as possible etiologic factors in pellagra. A possible transmitter of the disease was discovered in the stable-fly (Stomaxys calcitrans). Monkeys were subjected to repeated bitings of stable-flies which had previously bitten pellagrins but in none of the monkeys was the disease produced.[14c, 15a]

V. Numerous monkeys were inoculated with "filtered and unfiltered suspensions of material from pellagrins, including saliva, duodenal fluid, feces, tissues from the pharynx, stomach, intestines, brain, spinal cord, blood and lymph from the cutaneous lesions." By these methods the investigators were unable to transmit pellagra to the monkey.[15a]

VI. A condensed summary of the epidemiological studies of the Commission for the years 1912 and 1913 is as follows:

"1. The large active foci of pellagra in Spartanburg County were found in and near the large centers of population and particularly in cotton mill villages.

"2. (Age incidence. Sec. 10 under General Characteristics.)

"3. No definite connection between occupation and the occurrence of pellagra has been found, although high morbidity in women and children points to the home as the place in which the disease is usually contracted.

"4. In the group of incident cases most thoroughly studied, evidence of close association with a pre-existing case was disclosed in more than 88 per cent.

"5. A house to house canvass of the homes of over 5000 people, living in six endemic foci of pellagra, failed to disclose any definite relation of the disease to any element in the dietary.

"6. In the six villages, new cases of pellagra originated almost exclusively in a house in which a pre-existing pellagrin was living or next door to such a house, suggesting that the disease has spread from old cases as centers.

"8. Additional evidence has been obtained to support the conclusion that flies of the genus *Simulium* have nothing to do with pellagra.

"9. (Refers to animal inoculation and experimental study of intestinal bacteria, Sec. III, and V.)

"10. (Refers to studies on blood, Sec. I.)

"11. There is no evidence of inheritance of pellagra.

"12. The immediate results of hygienic and dietetic treatment in adults have been good, but after returning to former conditions of environment most of the cases have recurred. In children, prognosis is very much more favorable."[15a]

The most detailed studies were conducted in six of the mill villages (in Spartanburg County) in which pellagra was endemic and which were taken as representative of the surrounding country. The epidemiology of the disease was considered from every standpoint; in relation to race, sex, and age distribution; in relation to poverty; to occupation; to density of population; to location of domicile; to family history; to method of disposal of sewage; to household association; to diet; to previous state of health; to recurrence; to heredity; etc.[14a, 14b, 15a, 15c, 15d, 15f, 16b, 16c, 16d, 16e, 16f, 16h, 16i, 16j, 16k]

The field studies conducted during 1914 and part of 1915, according to the subsequent reports of the Commission, confirm the findings as given above. In the Third Progress Report[16] much confirmatory evidence is given to strengthen the conclusion of the Commission that methods of disposal of human wastes have a direct bearing on the spread of pellagra. The Commission leans strongly to the opinion that surface privies are in a large measure responsible for the spread of the disease. In support of this opinion, the Commission offers these facts:

(*a*) "Pellagra morbidity was higher in congested communities using surface privies than in more sparsely settled districts in which similar methods of the disposal of excreta were employed."[15d]

(*b*) "In the city of Spartanburg, the endemic foci of pellagra were located in districts in which surface privies were in use."[15d]

(*c*) "Since the installation of the sewer system (at Spartan Mills), the spread of pellagra has been decidedly restricted. In 1916, the only considerable focus of new cases was in the immediate neighborhood of a dilapidated out-house privy used by pellagrins."[16j]

Goldberger's Work.—The fact that in institutions where pellagra was either epidemic or endemic nurses and other attendants appeared uniformly immune was to Goldberger's mind a flat contradiction of the communicability of the disease. Epidemiological studies in a number of such institutions brought to him the knowledge that a difference existed in the diet of the inmates and in that of officers and attendants. He found the inmates on a diet which impressed him by the fact that it contained a disproportionately small amount of animal protein food and a disproportionately large part of the vegetable component. Legumes were inconspicuous.

To Goldberger this "very strongly suggested the idea that the disease was dependent upon a diet that was for some reason faulty and that this fault was in some way either prevented or corrected by including in the diet larger proportions of the fresh animal foods."[20]

A practical test of the worth of this idea was decided upon. The institutions selected for the experiments were two orphanages at Jackson, Miss., and the Georgia State Sanitarium, each one an epidemic focus of pellagra.

The chief modifications in the diet at the orphanages, put into effect by Goldberger on Sept. 15, 1914, were as follows: Fresh animal proteins were increased by giving to each child 14 oz. of milk and one egg per day and by serving fresh meat three or four times per week; leguminous proteins were increased by serving beans and peas with nearly every midday meal; wheat bread instead of cornbread in many of the meals; cane syrup was excluded for some weeks, after which it was allowed in limited amounts; oat-meal instead of grits was served at the morning meal.

In the two orphanages, during the spring and summer up to Sept. 15, 1914, the combined cases of pellagra numbered 209; of which not less than 23 had had at least two annual attacks. One hundred seventy-two of the cases completed the anniversary of their attacks under the observation of the investigators (Goldberger and Waring). There was but a single recurrence. Without dietary interference, the expected recurrences, estimated on basis of Rice's institutional cases, were placed at 86. No new case developed in either institution up to the close of 1915, at which time the diet test was no longer regarded as an experiment. In order that the test might not be weakened the sanitary and hygienic conditions, though regarded as poor, were not changed in any particular.*

At the Georgia State Sanitarium, Goldberger and Willets had supervision over the diet of the inmates of two wards. Here again was made a decided increase in the animal and leguminous protein foods. Of 36 colored female and 36 white female pellagrins, who were kept under observation until after the anniversary date of their previous year's attack, not a single one had a recurrence. Of the control group of 17 colored female pellagrins, 9 had recurrent attacks. Of the 15 white female pellagrins in the second control group, 6 had recurrences. The two control groups remained on the regular institutional fare and were kept under observation for a period of over one year.

In the early part of February, 1915, Goldberger organized *The Pellagra Squad* at the Mississippi State Penitentiary, which is located eight miles from Jackson, Miss. The main building occupies the center of over 3000 acres of land. There was no history of pellagra's developing on or being brought to this farm. On the offer of pardon at its termination, 12 men volunteered for the experiment. After keeping the squad on the regular prison fare for a period of two and one-half months without detecting evidence of pellagra in any of the men, the diet was changed at noon Apr. 19, 1915. From this time to Oct. 31, 1915, the men on the squad were fed the following articles only: "Wheat flour (patent), corn meal, grits, corn starch, white polished rice, standard granulated sugar, cane syrup, sweet potatoes, pork fat (fried from salt pork), cabbage, collards, turnip greens, and coffee." An average of 3.32 lb. of food, representing 2952 calories per man per day, was allowed. One of the volunteers was dismissed on account of physical disability shortly after the squad was organized. Of the remaining 11 men, not less than six had developed symptoms justifying a diagnosis of pellagra at the termination of the experiment, Oct. 31, 1915. Of the 30 convicts who remained at the farm throughout the time of the experiment, but who ate the regular prison fare, not one showed "even a suspicion of the disease."[21]

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* The writer visited the larger of these institutions in March, 1917, and again in July, 1917. He was told that Goldberger's menus were still being followed as closely as the resources of the institution and the increased cost of food would permit. Not a single case of pellagra was discovered after an inspection of all the juvenile inmates, both old and new admissions.

Goldberger next determined on an experiment calculated to test the infectivity of pellagra. Harris's[22] report of the successful inoculation of a monkey with pellagra has not been confirmed by similar experimental attempts on the part of other investigators.[15a, 23, 44]

In order to strengthen the value of his experiment, Goldberger conceived the daring idea of testing the infectivity of pellagra "on an animal species known to be susceptible, namely, man himself."

Of the 20 brave individuals who volunteered for the experiment, 16, including one woman, were selected. Their ages varied from 26 to 42 years.* They continued customary habits of life and diet, and were not confined in any way during the time of the experiments. Experimental materials were obtained from 17 cases of pellagra of various types and different grades of severity, including three fatal cases. The materials used were blood, nasopharyngeal secretions, epidermal scales from pellagrous lesions, urine and feces. "Blood was administered by intramuscular, or subcutaneous injection; secretions by application to the mucosa of the nose and nasopharynx; scales and excreta by mouth. Both urine and feces were ingested by 15 of the volunteers, five of whom also took blood, secretions, and scales. . . . During a period of between five and seven months, none has developed evidence justifying a diagnosis of pellagra."[25]

For the periodic recurrence in pellagra, Goldberger has offered the explanation that, as in beriberi, changes in diet are brought about by changes in season.

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* The criticism offered that the experiment deserves no weighty consideration because it was made on men in the active period of life when there is a recognized resistance against pellagra will, in the writer's opinion, appeal to those already inclined to accept the infectious nature of pellagra rather than to those with an open mind.

For the frequent development of pellagra in the insane in even well-provisioned institutions and homes, he offers an explanation by calling attention to the fact that eccentricity in taste is common in the insane, who sometimes refuse to eat at all.

He thinks, moreover, there are enough instances of eccentric taste among even the well-to-do to account for the development of the disease at times in this class.

Goldberger has very carefully worded each one of his articles bearing on the relation of a deficient diet to pellagra. Any misinterpretation or any confusion of the terms, deficient and insufficient, would at this time seem inexcusable. The writer, however, believes that in the minds of a large number of medical men such confusion still exists. The term "deficient," as Goldberger insists, when applied to diet has "a special, technical and restricted meaning."

Vitamins.—Funk advanced the hypothesis that there exist a number of chemical substances (vitamins), the absence of any one of which from the diet would cause abnormal metabolism; this abnormal metabolism would result in one case in beriberi, in another scurvy, in another rickets, and in another pellagra, according to whichever one of the vitamins was absent.

These four diseases have come then to be regarded and called the deficiency diseases or again diseases due to deficient diet.

It is in this sense that Goldberger uses "deficient diet."

McCollum and his coworkers claim that Funk is in error in believing that there are a number of these specific protective substances; that there exist but two such substances, fat-soluble A and water-soluble B; the absence of the former from the diet causing beriberi, the absence of the latter causing xerophthalmia. By experiments on guinea pigs they have proven to their own satisfaction that scurvy should not be classed among the deficiency diseases. They believe that scurvy, rickets and pellagra can be accounted for in a satisfactory way without assuming that there exists for each some unknown protective substance or vitamin. They will attempt to show that each of these three diseases is due to "unfavorable proportions among the well-recognized constituents of diet as well as of the two but recently appreciated ones, together with unsatisfactory physical factors and injury wrought through the agency of microörganisms inhabiting the alimentary tract."

A forthcoming paper discussing pellagra from an etiologic standpoint is promised by them.

SYMPTOMS

Following the appearance of pellagra in this country in epidemic form, fulminating types of the disease were not infrequent. The number of such cases has gradually decreased, so that at the present the foudroyant type is rarely seen. Indeed, pellagra is now regarded as being essentially chronic in nature.

The first season of its attack may leave little imprint on the victim. Content with this opening, the disease lies dormant for a period, during which the patient regains *almost* normal health. With the recurring season, a more vigorous attack is made. When next the disease recedes, the pellagrin has suffered too great a loss in energy to permit the regaining of full health.

The clinical picture of pellagra, then, is one in which a gradually increasing disease-force seasonally attacks a victim with a gradually decreasing resistance. The fight is not continuous but it is renewed season after season for several or even many years, until the resistance and the vitality of the pellagrin are spent. This, eventually, is hastened by any concomitant or accidental disease.

Such is the history of pellagra in the adult.

The symptomatology of the disease in adults is characterized by marked and wide variations.

In one class of cases, generally mild ones, skin manifestations appear first, and may predominate throughout the course of the disease.

In a very large class of cases, digestive symptoms appear before either skin or nervous symptoms. If there exists a concomitant uncinarisis or an amebiasis, gastro-intestinal symptoms may continue more distressing than any which develop subsequently.

In still another class, nervous or mental symptoms are observed before either cutaneous or digestive manifestations.

By no means, however, should it be considered that the first-appearing symptoms will determine the course of the disease with reference to which group of symptoms, skin, digestive, or nervous, is to be preponderant for that case.

Skin symptoms frequently give way in importance to other and graver ones. Psychic disturbances may be marked, yet the pellagrin dies from the exhaustion due to an intractable diarrhea rather than from brain or cortical lesions.

Again, in the same patient, one group of symptoms will predominate one season, while another group will predominate another season.

Pellagra in childhood ofttimes presents a very different picture from the adult type which has been thus briefly sketched.

In the pellagra of childhood, skin manifestations are always the earliest recognizable symptoms of the disease. In a very large proportion of pellagrous children, cutaneous symptoms remain the most marked and important throughout the course of the disease. There may be justification for the discussion of the condition, *pellagra sine pellagra*, in the literature of the disease referring to its occurrence in adults but such a condition in no way pertains to the pellagra of childhood.

The writer in a paper presented to the Section on the Diseases of Children, A. M. A., June, 1912, called attention to the mildness of pellagra as observed in some children and suggested the probability of such being a rather frequent occurrence. Since then abundant confirmation of the above observation has been found in the literature of the older writers: in the writings of Sambon, Wood, etc.; in the reports of various pellagra commissions, and also in the writer's subsequent and broader experience with the disease.

Case after case of pellagra in children has been personally observed in which there existed abundant, definite, and marked skin lesions without any other pronounced evidence of disease or of illness.

The following cases illustrate the mildness of the disease as it frequently occurs in childhood:

M. McG., girl, age 11 years; B. McG., girl, age 8 years; W. McG., boy, age 6 years;—sisters and brother. Father and mother living and in good health. Dwelling, a neatly kept, clean, screened cottage in an insanitary neighborhood in which there exist, however, no open privies. City water supply. Father is a motorman who provides a good table but the children like neither milk, eggs, nor meat. The chief articles in the children's dietary are batter cakes and syrup, oatmeal and sugar, grits and gravy, fruits, especially apples and bananas, biscuits (wheat) and butter. All three children have typical symmetrical eruptions on their feet. The older girl has in addition a distinct though not a marked "collar." The younger girl's eruption extends above the ankles higher on the external surfaces of the legs than on the internal, a distinct line of demarcation; in addition she has a slight eruption on her hands; she has also a sore mouth, the mucous membrane in buccal region is congested, tongue is coated except on the edge, a small ulcer at either side of the labial commissure. The boy has an eruption on his hands which encroaches on the wrists, but his most marked eruption is on feet and legs (Fig. 148). All three children are happy, bright, and enjoy playing; they have no complaints; they give no history of diarrhea; they sleep well; they eat heartily of all articles of food that they like; all three belong to the fat rather than the lean type. The mother thinks the eruption is due to "weed poisoning," as all three children "broke out the same way last summer after the weeds got rank."

These children were not brought to the physician, but were accidentally discovered by him when making an investigation of a pellagrous family in the neighborhood.*

By the offer of prizes, the three children were induced to drink milk with each meal, to eat meat at least once per day, and to eat not less than four eggs per week. They have since learned to enjoy these articles of diet and they continue to eat a more liberal amount of animal proteins. They have been kept under observation for six months. As in many similar cases, every trace of their eruptions had disappeared within a few weeks after beginning the diet treatment. The mouth symptoms in the second girl had also disappeared in the same short period. It will be of interest to observe if a recurrence will take place in any of these children when next the weeds grow rank.

Skin Symptoms.—In the discussion of the symptoms of pellagra in this chapter, chief stress will be placed on the skin manifestations because such manifestations in children are not only the most important but they are frequently the sole appreciable evidence of the disease.

The erythema of pellagra appears suddenly. It looks very much like a marked sunburn, a condition for which it is frequently mistaken. There is usually some tension and swelling of the affected skin areas. Itching, if any, is slight.

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* A discussion of the pellagrous family referred to here will be found in an article in Medical Clinics of North America, May, 1918.

From the dermatologists has come the argument that since the eruption of pellagra passes through several stages, the term erythema is inappropriate, in that it is descriptive of only the earliest and the briefest of these stages. It has been suggested that the term dermatitis would be more accurately descriptive.

In the early or erythematous stage of the eruption, the line of demarkation between the normal skin and the affected skin, with the latter's tension, swelling and livid red hue, is most sharply defined. The redness disappears on pressure but quickly reappears when pressure is withdrawn.

An unfailing feature of the eruption is symmetry. Unless characterized by symmetry, no eruption need be considered in connection with pellagra. The erythema may appear on one member before it shows on the corresponding opposite member, but unless it shows on both within a few days, it is not pellagrous. The eruption may occur asymmetrically in isolated situations but it is always accompanied by a symmetrically arranged eruption in one or more of the usual locations.

Symmetry, moreover, is carried out not only as to location, but often in such exactness of size and contour as to give the appearance of duplicates.

The erythema reaches its height in several days or at longest a few weeks. After this the intensity of the redness is less noticeable. It no longer disappears on pressure. Soon the redness is changed to a brownish, bronze, chocolate, or plum shade, depending somewhat on whether the patient is of the blond or brunette type.

In the negro the eruption appears like soot-smears on the brown or less black skin. Later a grayish tint is added which does not efface the contrast between normal and affected skin.

After the erythema has passed its height and the redness has been replaced by one of the shades mentioned above, the skin becomes more or less roughened. Next exfoliation begins usually from the center of the eruption. At the periphery, a border of brown pigmentation persists for some time after desquamation is complete elsewhere. This border is often nothing more than an uneven, broken, or interrupted line, but it very distinctly defines the limit of the previous erythema. This line is known as the *hyperkeratotic border* and often proves a valuable diagnostic aid in cases in which exfoliation has taken place. When the border has width, the edge toward the previously existing erythema is serrated and of a deeper brown color. The distal edge is not marked but is lost in the normal skin.

After exfoliation, in first attacks the skin looks smoother and softer than the skin not involved. In contrast with the unaffected skin, the new skin is like that of a newborn infant. Gradually the new and the old skin meld and become indistinguishable except to the trained eye. Referring to aids in diagnosis between attacks, Roberts says the skin on the hands and arms, if previously involved, shows a mosaic mottling with cyanosis, when the arms hang loosely by the side. The same condition is present in the skin on the feet and legs when the patient sits. This phenomenon has been repeatedly observed in pellagrous children by the writer many months after their first and only attack.

Two or three months is the time usually covered by the eruption in passing through these various stages; erythema, pigmentation, and exfoliation. In some children, however, the writer has seen this cycle gone through within one-half that time.

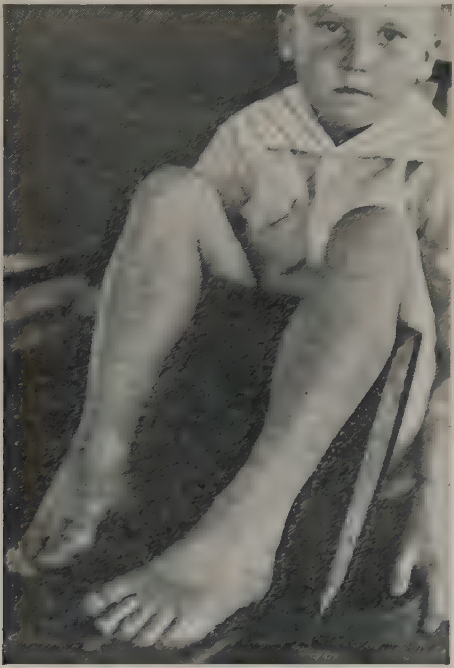

FIG. 148.—Eruption on feet, legs and hands. Erythema has subsided and desquamation has begun.

After repeated attacks the affected skin assumes a permanently scaly, roughened, and dirty appearance. A distinguished surgeon recently sent a child back from the operating-room to the ward of the hospital with a sharp injunction to the nurses in charge to never again bring into his presence an unbathed patient. Investigation revealed the fact that what the surgeon had taken for dirt was in reality areas of pigmented pellagrous skin on the child's feet, legs, and knees.

The description of the erythema or dermatitis of pellagra as given in the above includes only the "dry" form of the eruption. In order to prevent confusion in the reader's mind, nothing has been said up to this point about the "wet" form.

Superimposed on the erythema in some cases there are a number of vesicles or bullæ. When the vesicles rupture, there remains a raw, serous, sometimes blood-oozing base. Ordinarily this heals over rather quickly but occasionally infection takes place with ulceration into the deeper structure. The site of the "wet" eruption is the back of the hands, the feet, or the legs; sometimes all three. The patient presents a pitiful but revolting appearance. The prognosis in cases with the wet eruption is grave. This form of the eruption is rare in children, but it does occur.

The skin lesions of pellagra have a predilection for the exposed surfaces of the body. Observations based on many very mild cases, in addition to well-marked cases, lead the writer to the conclusion that in children the most frequent site of the eruption is the dorsal surfaces of the feet. Among the children in the South bare feet in great numbers appear in the early spring. In some communities many children do not wear shoes during any season. Children in whom the eruption is limited to the feet are rarely brought to the doctor. Seldom do they have symptoms sufficiently marked to attract attention. When pellagrous children are sought out, however, the medical man is impressed by the large number discovered who have but a slight though distinctly diagnostic eruption on the feet and nowhere else (Fig. 148).

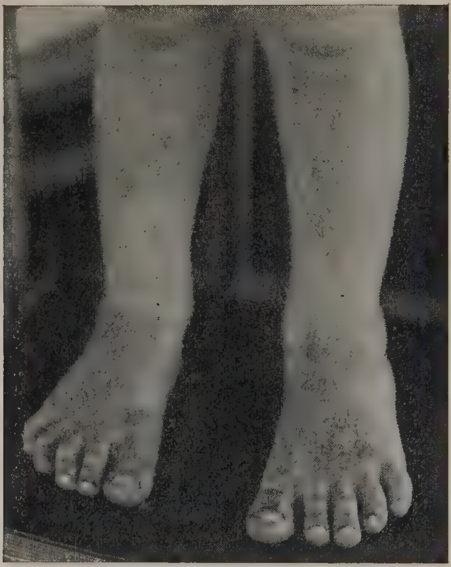

FIG. 149.—A frequent and often only location of the pellagrous eruption in children.

Cases which are brought to the physician are, as a rule, only such as have well-marked and multiple lesions. The hands and arms, the feet and legs, and the neck and face may all display the eruption. In well-marked cases, the eruption on the feet does not confine itself to the dorsal surfaces but extends from the toes to the malleoli, or even up the leg to the knee, forming the pellagrous "shoe" or "boot" according to the distance of the line of demarcation from the ankle (Figs. 149, 150). The heels and soles of the feet are rarely ever involved.

On the hands, the eruption occurs on the backs and usually extends several inches above the wrists. The terminal phalanges frequently escape, though the eruption is well marked over the remaining surface of the backs of the hands (Fig. 151). The palmar surface is rarely encroached upon. When the eruption appears on the forearm, it is usually limited to the extensor surfaces. The "pellagrous gauntlet" formed by the eruption on the hands, wrists, and forearms, with the line of demarcation encircling the latter midway between the wrist and the elbow, is a rather rare phenomenon in children.

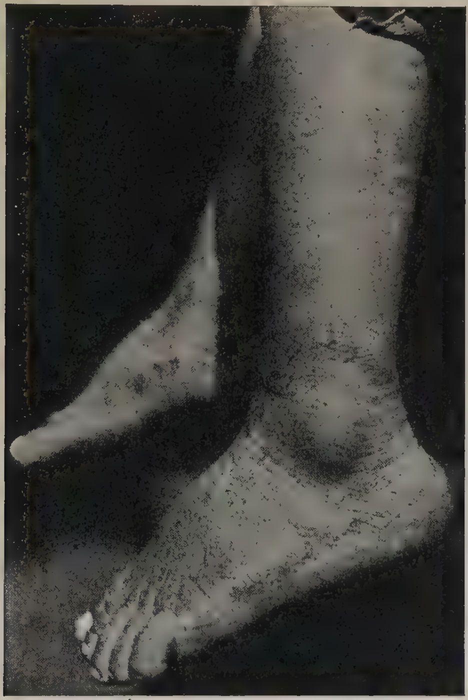

FIG. 150.—The pellagrous boot in boy age three years.

The neck is a more frequent location of the eruption in children than in adults. Equidistant from the midline on the back of the neck may appear spots of an eruption; or there may be a band extending around the neck, forming the "Casal collar" (Figs. 152, 153).

On the face, the eruption may also appear in spots on the forehead, cheeks, etc.; or it may be diffuse so as to form a "mask." In the latter

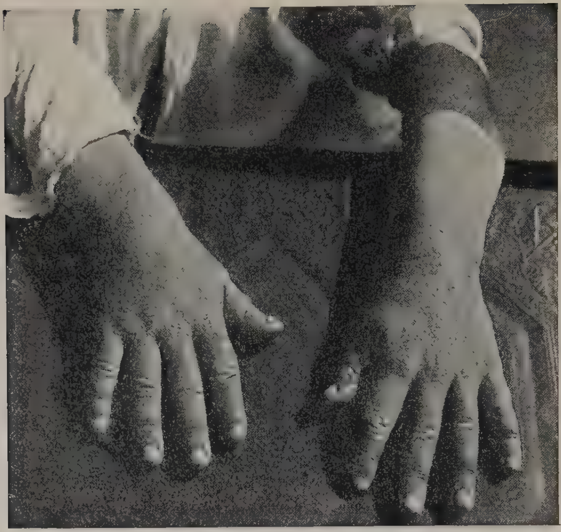

FIG. 151.—Typical eruption on hands and wrists of boy age three years. Pellagrous glove.

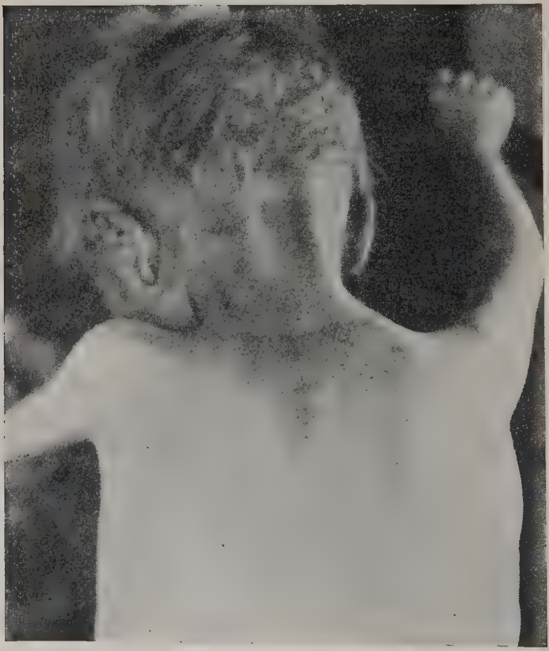

FIG. 152.—Casal's collar in boy age three years.

case, the only unaffected skin on the face is a narrow strip running parallel to the margin of the hair (Fig. 154).

In some cases the erythema spreads over the entire or almost entire surface of the body, *pellagra universalis.* This condition is very rare in adults, but in children that in warm weather wear only a thin one-piece garment and in boys that spend much time on the banks of the swimming-hole, a diffuse wide-spread eruption is not so rare (Figs. 155, 156).

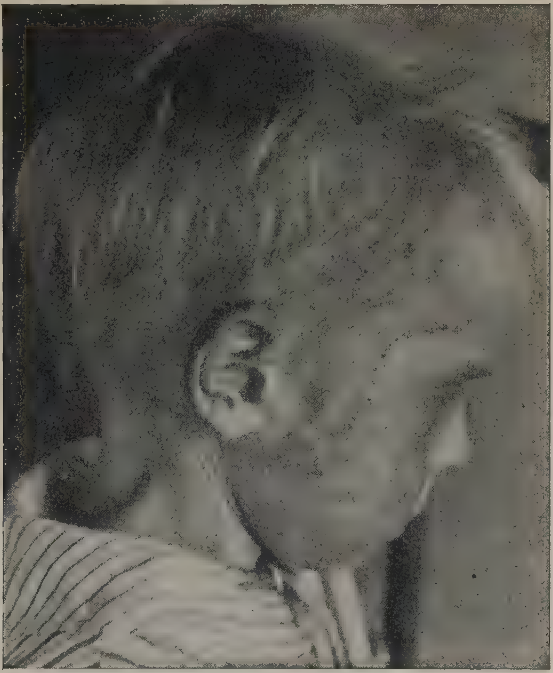

Fig. 153.—The hyperkeratotic border outlining pellagrous mask and Casal's collar, in boy age three years.

The extensive eruption frequently abates over a considerable portion of the body while still persisting on some of the more usual sites: as the feet, hands, face, neck, etc.

The severity of the disease is not necessarily more marked in children with the wide-spread eruption than it is in children in whom the lesions are confined to the usual locations.

For several years the writer has been interested in a class of cases occurring among nursing infants which, in some of its features, presents a clinical picture resembling pellagra.

As a rule these infants are only a few weeks old. They are covered with a universal erythema, superimposed on which, particularly on such locations as face and extremities, are numerous vesicles and pinpoint pustules. There is little or no elevation in temperature. The tongue is coated, but red on tip and edges. The tongue is moist. The entire mucous membrane in the mouth is fiery red, but there is no sprue visible. The stools are thin and frequent and of a brownish or greenish-yellow color. The infant is fretful, colicky, and sleeps poorly. The urine is voided freely and is colorless. There is a progressive but not a rapid loss in weight.

The most frequent diagnosis is syphilis, but such a diagnosis is contradicted by the family history, the Wassermann test, and the subsequent history. There is no great resemblance between the eruption in these cases and eczema, for which the condition is rarely ever mistaken. Investigation will generally reveal the fact that the infant is nursing a breast the milk of which, though secreting abundantly, is of very poor quality. Occasionally, it will be found that the infant is suckling a breast which furnishes an inadequate supply. In the latter event, it will also be discovered that the fluid intake of the baby has been supplemented by frequent administrations of sugar water, toddy, or catnip tea. When these cases are given proper nourishment the eruption and other symptoms disappear with almost magical rapidity.

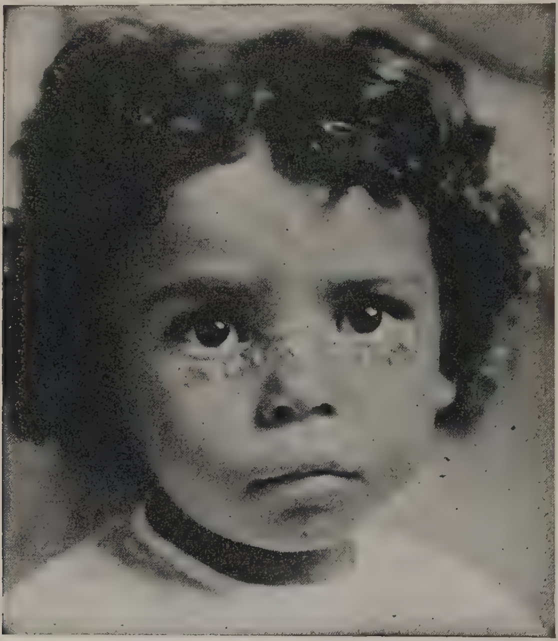

Fig. 154.—P. B., Male age two years. Mulatto. Showing pellagrous mask on butterfly.

The writer does not insist on classifying these cases as pellagra; he merely wishes to call attention to some points of resemblance. He believes, too, that until some more appropriate name is suggested, he is justified in applying to them the term *pellagroid*, in order to place them in a distinct class. They do not belong in the class with cases of acute inanition fever. In the latter, the fever is high, the urine is scant and stains the diaper; there is rapid loss in weight; the tongue is dry; there is constipation as a rule, and if there is a diarrhea, the stools are meconium like in color; there is no eruption.

Digestive Symptoms.—One of the most constant digestive symptoms of pellagra is inflammation of the mucous membrane of the mouth and tongue. In children, this inflammatory process is often so slight as to cause no complaint. Other children, however, are acutely conscious of the sensation of dryness, heat or burning in the mouth, so frequently complained of by adult pellagrins. These sensations may be felt not only in the mouth but in the throat, esophagus, and stomach. The buccal mucous membrane may be only slightly congested and roughened, or there may be marked stomatitis. Ulcers on the side of the cheek may be pinpoint in size or there may be large

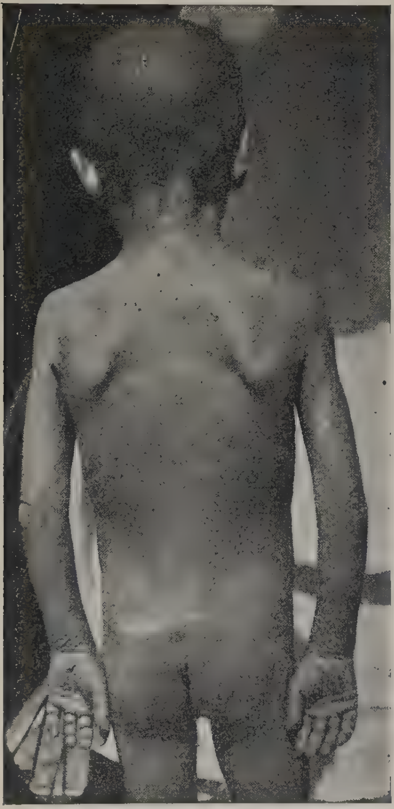

Fig. 155.—Pellagra universalis, in Italian boy who developed the disease after two years residence in this country.

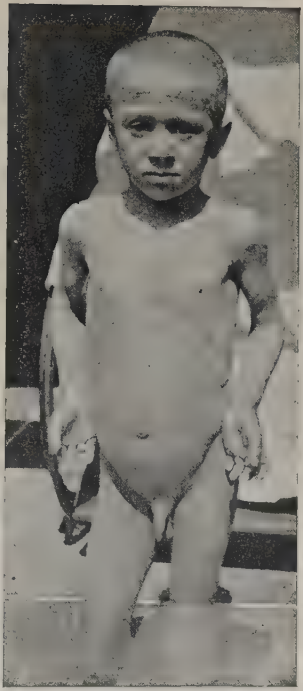

Fig. 156.—Front view of boy shown in Fig. 155. Note wrinkles on forehead giving aged appearance to child.

patches. The tongue may be coated except on the tip and edges, where it is fiery red with papillæ prominent. Later the whole-tongue may become congested and swollen. In adults, ulcerations in the mouth to any marked degree are uncommon except when preceded by a diffuse active inflammatory process. In children, a mild inflammation of these structures is sometimes quickly followed by widespread ulcerations (Figs. 157, 158). This is probably to be accounted for by the fact that, while adult pellagrins have, as a rule, sound teeth, juvenile pellagrins are frequently attacked at a time when the temporary teeth are in a process of decay. Ptyalism may be an annoying symptom. The gums may become spongy and show a tendency to bleed. The epithelial cells on the tongue sometimes are completely exfoliated. After the remission of acute symptoms, the tongue often appears smooth, glazed, and pale, the "bald" tongue, or the very red "cardinal" tongue.

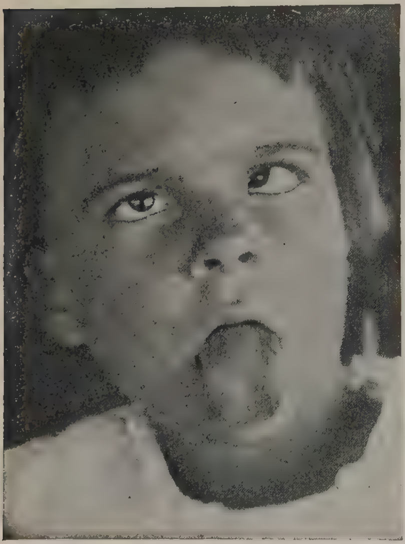

Fig. 157.—Pellagrous sore mouth in girl age six years. Also had eruption of feet, neck and spots of it on face.

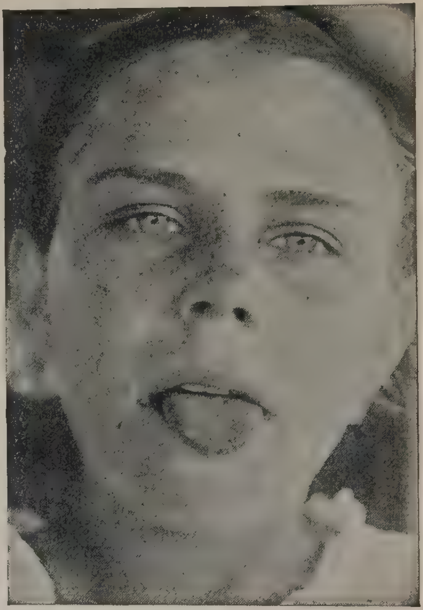

Fig. 158.—Pellagrous sore mouth. Girl age eleven years. Had eruption on feet and hands.

The appetite of adult pellagrins is usually greatly diminished, the patient complaining of a feeling of fullness—even with colicky pains—which bears no relation to the amount or the character of the food taken. It is hard to induce individuals possessing these symptoms to partake of a sufficient amount of food to stave off inanition. Other adults, however, overeat at times. Either extreme is rare in children with pellagra. The appetite as a rule is only slightly diminished. Little patients with rather severe stomatitis, gingivitis, and glossitis, who even suffer from gastralgia, have been known to partake of food with the zest of a healthy child. This fact unquestionably has some bearing on the more favorable course of the disease in children. There is no more impossible undertaking in the realm of medical endeavor than to cure pellagra in an individual who will not eat.

The diarrhea characterized by frequent, watery stools with peculiar bad odor, so common in adult pellagrins, rarely occurs in children. Not infrequently pellagrous children are constipated. If a diarrhea occurs, it is usually not so protracted as in adults. In very young children with pellagra, dysentery or ileocolitis is more common than in older children or in adults. It is doubtful, however, if the proportion of such conditions in these very young pellagrins is much greater than in non-pellagrous children of the same age who live in similar environments.

Nervous and Mental Symptoms.—Nervous and mental symptoms of pellagra in children are comparatively insignificant. With the exception of one case, a boy five years old, in whom the lower tendon reflexes were definitely exaggerated, the writer has been unable to elicit nervous symptoms in pellagrous children. Pain in the back, a common symptom in adults, has been complained of by no child pellagrin known to the writer. Pressure has failed to elicit tenderness along the vertebral column. As already stated, many of the author's cases have been exceedingly mild. No death from pellagra has occurred in his practice. He is, therefore, unacquainted with the severe forms of the disease in children. Children occasionally complain of burning or even pain in the stomach, but it is very doubtful whether such symptoms are due to disturbances of the nervous system. It is more probable that they arise from inflammatory processes *in situ.*

Headaches, vertigo, ringing in the ears, etc., have never been complained of by any pellagrous child known to the writer.

The mental condition of the pellagrous child, as a rule, is unimpaired. Several children in the writer's series of cases have been apathetic during the active period of the disease. A little girl three years old, who has been under observation for six months, is inclined to tears without provocation. Imaginary slights are frequently cherished. Whenever the nurse or the doctor passes her without bestowing what she regards as sufficient attention, she bursts into tears, not crying aloud but sobbing in a most heartbroken fashion. This child will rarely play games with the other children in the institution but is usually found sitting to one side watching the others. She neither livens up nor gladdens up, though all other evidence of pellagra has disappeared. Other children in the institution have come to realize how anxiously the attendants watch for the evidence of a final cure, and there are days when the physician is greeted at the door by one of these other children with the tidings—"Helen smiled," or "Helen laughed today." This is the only case known to the writer of a child pellagrin's mental condition showing impairment.

DIAGNOSIS

There is little likelihood of mistaking pellagra in a child for any other condition. The disease is frequently overlooked in children because of its mildness. The disease cannot be diagnosed without the presence of skin lesions, and when the latter are present they are sufficiently characteristic to render a diagnosis easy, provided the possibility is borne in mind. The skin lesions of pellagra are as definitely typical and, therefore, diagnostic as are the skin lesions in scarlet fever, measles, chicken pox, etc.

There are no laboratory aids to the diagnosis of pellagra.

PROGNOSIS

The reader has, no doubt, already correctly inferred from what has been said in the discussion of symptoms that the prognosis in pellagra in children is much better than in adults. When the fulminating type of the disease was not of infrequent occurrence, a number of children died from pellagra; but fatalities among children are now exceedingly rare. It must be added, however, that should typhoid fever, pneumonia, scarlet fever, or any other illness of a serious nature befall the pellagrous child, both the intercurrent disease and the pellagra may assume a grave nature.

Prognosis as to recurrence depends much on how close the observation and the control of the case may be. Whenever it is reasonably certain that proper diet and proper hygienic care will be continued, it can safely be predicted that no recurrence will take place. On the other hand, a recurrence can be expected in about 50 per cent. of the cases in which the patients relapse into old habits and customs relative to the diet and hygiene.

PATHOLOGY

The pathological changes produced by pellagra either in the skin, in the gastro-intestinal tract, or in the central nervous system are neither constant nor characteristic. Morbid changes of a pronounced nature are found but these changes so vary with the symptoms and the chronicity of the disease that it is impossible to distinguish in the postmortem picture the essential from the non-essential.

Postmortem studies are still further confused because such studies necessarily are frequently conducted on those dying from a disease intercurrent with or complicating pellagra.

Lynch[44] states that the most constant postmortem findings in pellagra are those referable to the intestines and consist of morbid changes characteristic of colitis. He claims these changes are found in both juvenile and adult pellagrins and that furthermore they take place even though diarrhea has not been a feature of the disease.

TREATMENT

The first step in the treatment of the pellagrous child is to ascertain the existence or non-existence of any concomitant disease. A cure or even improvement of pellagra should not be looked for in a child infected with hookworms, malaria, or amebæ. Moreover the concomitant disease is, as a rule, the only one which demands treatment by drugs.

Whenever possible, the child should be removed from the environment in which pellagra developed. This is rarely feasible in the class of children in which the disease is most commonly found. However, the rural and backwoods people are fast losing their superstitious fear of the hospital. Each year witnesses an increasing number of these families which are willing to send their sick to state or county institutions for treatment. State officials must now be educated to a realization of the fact that the backwoods people are given to snuff-dipping, to moonshining, to feuds, etc., more because of their diseased bodies than because of depravity; to a realization of the fact that these people are inefficient, thriftless, and "trifling," not so much from inborn laziness as from devitalizing disease forces. The state must be educated to a realization of the wisdom and the economy of seeking out and eradicating hookworm, amebic dysentery, malaria and pellagra. When the state is as willing to enforce health as she now is to enforce prison sentences, there will be fewer occasions for imposing the latter.

If not feasible to remove the patient from his environment, the physician should, at least, make an endeavor to improve that environment by calling attention to insanitary and unhygienic conditions and by pointing out means and methods for their correction.

No matter how divergent may be the views relating to the etiology of the disease, all who are familiar with pellagra admit that an improved and regulated diet is of the greatest importance in the treatment.

The writer has come to rely more and more on the efficiency of animal proteins, such as are contained in milk, meat and eggs, as curative agents in the management of these cases in children.

Goldberger's diet included, in addition to the above, legumes such as fresh or dried peas and beans. To the inclusion of these articles in the diet for pellagrins in general the writer offers no objection. He heartily indorses Goldberger's propaganda to promote a wide-spread planting and cultivation of these leguminous vegetables. In the treatment of pellagra in children, however, the writer has frequently experienced difficulty in having these vegetables prepared in a way to appeal to the child's taste and at the same time to be suitable to his digestion. He has gradually come to the point of dropping legumes from the list of essential foods in the treatment of the disease in children.

The writer does not advocate the discontinuance of cautioning the pellagrin against exposing himself to the action of the sun's rays, but he wishes to relate that some of his cases who have played bare-headed and almost bare-bodied in the sun have yet improved more rapidly and satisfactorily than did many of his former cases which were kept indoors for too long a period. Confinement in the house frequently interferes with the child's appetite and general well-being and results in more harm than good. As a protection against the action of the sun's rays, zinc oxide ointment applied fairly liberally over the sites of the eruption has proven satisfactorily effective. On the skin protected in this manner the writer has never observed any marked ill effect from sun exposure of reasonable length.

The writer does not advocate the continuance of corn meal or corn products in the dietary of the pellagrin, but he does insist that after eating an egg or a piece of meat or after drinking a glass of milk it is better to allow the pellagrous child to "fill up" on corn bread, if there is nothing more suitable at hand, than to go hungry.

Unless the physician in charge has had experience and unless he has profited by his experience, the pellagrous child is very likely to be overtreated. Frequently such a child will be found taking arsenic, nitric acid, bismuth and opium, and several other kinds of medicine, when the only real needs of the child are proper hygienic care and proper food.

The hygienic care consists in cleansing baths, clean clothes, cleanly habits, fresh air, a limited time in the open, sufficient rest, proper exercise, and proper toilet of the mouth.

The feeding care has to do not only with the selection of proper foodstuffs but also with the proper preparation and cooking of that food.

Symptoms which require special treatment are stomatitis and diarrhea. The services of a dentist should be sought for any defective teeth. The mouth should be cleansed of all food debris after eating. As a local application to the stomatitis or ulcerations nothing has proven of more value than a weak solution of silver nitrate. This can be applied daily so long as the condition persists.

There are no drugs which seem to exert any influence in controlling the diarrhea. Fortunately this condition, as before stated, is rarely ever severe or protracted in children. Rest in bed with especial care to the diet will ordinarily result in a prompt improvement.

One of the first indications of improvement is a gain in weight.

Before closing this chapter, the writer wishes to insist that the pellagrous mother should not nurse her infant. This position is taken not for the reason that the writer believes in the possibility of transmitting the disease in this way but for the reason that a pellagrous mother is not capable of secreting a good milk. Furthermore, the tax on her system incident to lactation will influence unfavorably the progress of the disease in the mother.

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CHAPTER XXXII

RICKETS

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Rickets is the most common chronic nutritional disease occurring among infants of the temperate zone. It is found more commonly among the infants of the large cities, but is by no means absent in the rural districts. It is manifested particularly in alterations of the bones, especially of the growing portions of the long bones, the epiphyseal ends. For many years it was regarded merely as a disease of the bony system but in recent years it has been realized that this viewpoint is too narrow and that the disorder is systemic, involving the muscles, nervous system, blood and, in fact, almost all the organs and tissues of the body. It is, however, a disease especially affecting the growing bones and, therefore, occurs most frequently among infants during the first year or two of life. In itself it is responsible for few deaths, but the constitutional weakening which it occasions results in secondary infections, and causes it to play an important rôle in the infant morbidity of our large cities.

History.—Whether rickets should be considered a modern disease, or whether it was equally prevalent centuries ago, has been a subject of much discussion. Statements have been found in writings of the Greek philosopher Plato, and of the Roman physicians, Soranus of Ephesus and Claudius Galenus, which seem to refer to this disorder. These descriptions are not, however, sufficiently precise to convince one that they do not have reference to some other diseases accompanied by deformity of the bones.

The most remarkable feature in relation to the history of rickets is its sudden appearance, or at least, marked increase in incidence at the beginning of the seventeenth century. To such an extent was this manifested in England that a commission was appointed, with the celebrated Glisson at its head, to gather reports and communications relative to its incidence. This report of Glisson and his collaborators, especially the second edition which appeared in 1660 under the title "De Rachitide," forms the foundation of our knowledge of rickets and was translated from Latin into many languages. On account of this early description writers on the Continent termed and still term rickets the "Englische Krankheit."

In the eighteenth century many important works are to be found on this subject; most of them, however, confuse scurvy, bone tuberculosis and syphilis with rickets. Among the writers of the nineteenth century, who aided in a better understanding of this disorder, may be mentioned Trousseau and Broca in France, Virchow in Germany and Kassowitz in Austria.

During the last few years interest in rickets has been revived due to the discovery of vitamins and the fact that some have associated the etiology of rickets with a lack of one of these unknown factors. It has been greatly stimulated also by the development of experimental methods which enable us to bring about with regularity in animals a disorder accompanied by pathologic lesions identical with those seen in infantile rickets.

Distribution and Frequency.—Rickets occurs most often and in greatest intensity in the temperate zones. Probably no country is entirely free from it. The reports of its occurrence in the Far East and in uncivilized lands are, however, too meager to permit of more than general deductions. Jeffery and Maxwell* state that "the most characteristic anomaly of infant disease in China is the scarcity of rickets." However, in an article published a few years later, Harston† writes that "amongst native children rickets formerly was very rare; now it is of frequent occurrence, especially in South China." It is of interest to learn of its comparative rarity in large cities such as Tientsin and Peking. In Srinagar (Kashmir) there is almost no rickets in spite of the fact that the children are nursed two to three years. Hirsch‡ reports a similar low incidence in India, and Baginsky, in the course of his travels in the East, noted a similar paucity of cases among the Arabs, Bedouins and Buchars. It is evident that the common assertion that rickets is a disease of the poor, holds good only for certain countries and for people living under peculiarly unfavorable conditions.

It is not possible to state the incidence of rickets for any particular country, nor to compare this incidence between the rural and urban population. In regard to rural children there are no figures on which to base a statement, although it is the general experience that this disorder occurs more frequently in cities, especially in the large industrial centres. Thomson§ is of the opinion that more than 50 per cent. of the infants in Edinburgh, London, Glasgow and Manchester, show unmistakable signs of rickets. I believe that at least this percentage holds true for New York City and for other large cities in the United States. Were careful clinical examination and chemical analyses of the blood employed to establish the diagnosis and to discover the disorder in its earlier stages, the number of cases would be considerably higher. The percentage of incidence will depend also upon race. As is well known, Italian and negro babies in our large cities are especially prone to rickets. Many years ago Snow reported that in spite of the fact that the Italians were better fed and clothed in America than in their native land, and that they nursed their infants in the great majority of cases, rickets developed in about two-thirds of their offspring. The same susceptibility is evinced among the negroes in our large cities. A few years ago, in the course of an investigation of the infants in a negro district in New York City, Hess and Unger* found that fully one-half of the breast fed babies, and two-thirds of the artificially fed, showed definite clinical signs of rickets. This high susceptibility must not be attributed solely to race, for these same people are almost free from this disorder in their native land. An inquiry among the physicians of the islands of the West Indies, whence most of these negroes emigrated, brought unanimous reports that rickets was almost unknown in these islands.† Vipond examined 200 colored children in Jamaica and found rickets rare among the colored children in Kingston. This seems to be true also of the African negro. In a paper on rickets in German East Africa, Peiper writes that there is no rickets among the negroes, and that there is no reference to this disorder in the medical reports of the colony from 1903 to 1911. Such‡ a statement gains added interest when we learn that it is the custom among these people to feed infants from the first day of life with a thick cereal pap. It would be of value to ascertain the incidence of rickets among the negroes of the southern states of this country, and particularly of its relative distribution among the urban and the rural population; there are, however, no figures available bearing on this aspect of the subject.

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* Jeffery, W. H., and Maxwell, L. J., "Diseases of China, etc." P. Blakiston and Co., Philadelphia (1910).

† Harston, G. M., "The Care and Treatment of European Children in the Tropics." William Wood and Co., New York (1913).

‡ Hirsch, A., "Handbook of Geographical and Historical Pathology." London, New Sydenham Society (1885).

§ Thomson, J., "Clinical Studies and Treatment of Sick Children," 3d ed. Oliver and Boyd, Edinburgh (1921).

The occurrence of rickets varies according to attendant circumstances. This was strikingly demonstrated during the World War, when it blazed forth in almost epidemic proportions in Europe. We have a mass of reports from Germany, Austria and Belgium recounting its marked increase and unusual severity during this period. Some of these no doubt refer to nutritional conditions which were not true rickets, and many were complicated by other disorders which affect the bones. In the United States, the consensus of medical opinion seems to be that rickets is steadily becoming less frequent. In my opinion there has been a decrease in New York City during the past 20 years.

Seasonal Incidence.—A remarkable phenomenon noted by Kassowitz years ago is the striking seasonal incidence of rickets—its increased occurrence and intensity in the early spring, and tendency to fall to its lowest level in the late summer or autumn. Not only the clinician, but the pathologist as well, is aware of this seasonal augmentation and diminution. Schmorl's study of 386 cases (Fig.

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* Hess, A. F., and Unger, L. J., J. A. M. A., lxix (1917), p. 1583.

† Hess, A. F., and Unger, L. J., J. A. M. A., lxx (1918), p. 900.

‡ Peiper, O., Veroeffentlich. d. Medizinalverwaltung, Berlin, xi (1920), p. 6.

159) brings out this aspect admirably. Pellagra, tetany, keratomalacia, and to a less extent, beriberi, are associated with a similar seasonal variation. The marked increment of rickets in the spring was attributed by Hansemann to prolonged indoor confinement of the infants throughout the winter, and was the basis of his "domestication" theory. By some, it has been attributed to a change in the milk due to an alteration in the fodder of the cows. However, in a recent study Hess and Unger* found that dried milk from pasture-fed cows was unable to diminish the incidence of rickets in the winter. In view of the marked effect of sunlight—a subject which is fully discussed in subsequent chapters, it is clear that hygienic factors, especially sunlight, and not dietetic factors, play the dominant rôle in the marked seasonal variation of this disorder. *There is not merely a seasonal incidence of rickets but a definite intraseasonal variation.* The cases begin to be apparent in November, increasing in number and severity month by month until the peak is reached about the middle of March. A statement of maximum incidence in any locality in the temperate zone should, therefore, be based on examinations carried out during the month of March.

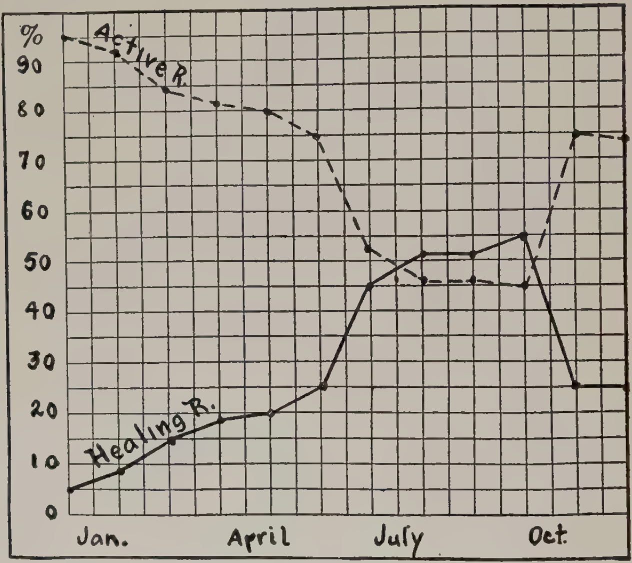

FIG. 159.—Result of microscopic study of the bones of 386 consecutive necropsies on infants; marked decrease of active rickets accompanied by marked increase of healing rickets during the summer months.

Age of Occurrence.—Rickets is encountered clinically most often during the second half of the first year and the first half of the second year of life. Cases, however, are not infrequent at five or six months of age, especially in premature babies or in twins. Probably it will be found that rickets, like scurvy, occurs earlier than has been supposed, and that in its milder stage it is merely latent and not subject to ordinary methods of diagnosis. Most of the rickets observed after two to three years of age is to be regarded as the vestiges of this disorder rather than as an active pathologic process. Among the negroes, however, the disease is not only of greater intensity, but is prolonged into a later period of life than is the case among white children.

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* Hess, A. F., and Unger, L. J., Am. J. Dis. Child., xxiv (1922), p. 327.

A generation or more ago **congenital rickets** was considered not only well established but of frequent occurrence. In this respect the history of rickets has been similar to that of tuberculosis, in that today a congenital case of either disease is considered a rarity. As far as I am aware, no instance of congenital rickets has been substantiated by histologic examination; Schmorl failed to find any in his pathologic studies. Formerly osteogenesis imperfecta, achondroplasia and hereditary syphilis were regarded as congenital rickets. Craniotabes was considered pathognomonic of rickets. Were we to accept this point of view, congenital cases would be by no means rare. It is true that some beading of the ribs is frequently found in the newborn, but the costochondral junctions do not show the characteristic pathologic changes.

Late rickets is a form of this disorder which always has excited great discussion ever since Ollier described it in 1861. In many cases the diagnosis has been questioned; furthermore, doubt has been raised as to whether the cases should not be considered rather as instances of a prolongation, or recrudescence of infantile rickets. Some have included under this term not only cases with typical signs, but also the scoliosis, flat foot, genu valgum and other mild deformities which make their appearance so frequently in later childhood. The consideration of late rickets must be regarded from a new point of view since the period of the World War and the year following the armistice. As the result of the privation occurring in the Central Empires during these years, late rickets suddenly became a common disease. It will be well, therefore, to base this brief survey on the recent descriptions of German and Austrian physicians. The signs and symptoms are, in general, those which will be described below in connection with infantile rickets—beading of the ribs, enlargement of the epiphyses and characteristic radiographic changes at the ends of the long bones. A frequent early symptom was a waddling gait, and, chiefly among girls, pain or tenderness of the ribs, of the spine or of the pelvis. Later genu valgum or varum often developed. The period of greatest incidence was that of secondary growth, the years 14 to 17. Heavy work, especially lifting, seemed to act as an exciting cause.

There are observations recurring in these descriptions which are of broader significance. For example: The marked increase of infantile rickets, as well as of osteomalacia throughout this period, constitute evidence in favor of common etiologic factors in these two conditions.

The reports of osteomalacia accompanied by involvement of the nervous system, such as an increase of the deep reflexes, and of mechanical and electrical hyperexcitability of the superficial nerves, call to mind the tetany which so frequently accompanies infantile rickets. The consensus of opinion as to the specificity of phosphorus and cod-liver oil is also of interest in this connection. It is probable that in many instances the disorder was not simply late rickets, but that partial starvation led to osteoporotic changes in the bones. In this way the frequent occurrence of fractures may be explained. However, as we shall point out below, osteoporosis and fractures occur in infantile rickets more often than has been supposed.

Etiology and Pathogenesis.—The present is the most eventful period in the history of rickets since the seventeenth century, when the disorder was first described. This is due largely to the fact that rickets can now be regularly produced and controlled in animals. Ever since experimental methods have been employed in the investigation of medical problems, there have been attempts to induce rickets, but until recently the technique has not been perfected sufficiently to render the experiments reliable and constant. The first successful experiments of this kind were those of Mellanby* who made use of puppies for this purpose. He was able to formulate rachitic as well as antirachitic dietaries, and employed histologic and chemical examinations of the bones and radiographs of the epiphyses to establish the diagnosis. Although his results were not absolutely constant, they constituted a definite step in advance. More recently Sherman and Pappenheimer,† as well as McCollum, Simmonds and their associates‡ have devised diets which can be so regulated as to produce, or fail to produce, rickets in rats. For these experiments young rats have been used about four weeks of age, weighing about 40 gm. The bony lesions in these animals closely resemble both radiologically and histologically those characteristic of the rickets of infants. It was found that where the phosphate or the calcium content of the diet was low, rickets resulted in a period of about four weeks. The information which animal experiments have furnished in the short period during which they have been employed is most promising and will be referred to again in connection with the pathology of human rickets. One of the main points which they have established is that heretofor too great emphasis has been placed on the calcium factor in the etiology, and too little on the phosphorus ion. It is increasingly evident that within the next few years experimental rickets is destined to elucidate many questions, especially in regard to etiology and pathogenesis, which it has been impossible to unravel as long as they were enmeshed in a confused mass of clinical observations. The question has been raised as to the identity of rat rickets and human rickets. It would seem that the parallelism of the pathologic lesions, both gross and microscopic, furnish sufficient evidence for us to consider them identical. This does not, however, warrant the inference that the metabolic processes which occasion these lesions are in all respects the same. It certainly does not indicate that rickets develops in infants as the result of dietary deficiencies similar to those in the standard rickets-producing diets. Dietetic conditions are far more complex in the clinic than those which have been employed in the laboratory. For example, although rachitic infants may suffer from a lack of phosphorus in their tissues, this is not due to such a simple phenomenon as a deficiency of phosphorus in their food, but results from secondary factors influencing absorption and metabolism.

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* Mellanby, E., Medical Research Committee, Report No. 38, London (1919).

† Sherman, H. C., and Pappenheimer, A. M., Proc. Soc. Exp. Biol. and Med., xviii (1921), p. 193.

‡ McCollum, E. V., Simmonds, N., Parsons, H. T., Shipley, P. G., and Park E. A., J. Biol. Chem., xlv (1921), p. 333.

Rickets has been ascribed to almost every deleterious influence which is known to affect human beings—to defective hygiene, improper diet, intestinal toxins, bacterial infection, derangement of the secretion of the endocrine glands, etc. Moreover, these influences have been supposed by some to exert their harmful effect on the parents and the disease was thought to be transmitted to the infant through heredity, by others to be intra-uterine or prenatal in origin, whereas the majority are of the opinion that the main etiologic factors play their part in postnatal life. Discussions covering this wide field have been carried on in the medical literature ever since the days of Glisson, but have assumed a scientific interest and importance only since rickets can be studied experimentally in animals.

Those who support the theory of *heredity*, more especially Siegert,* gathered statistical evidence showing the marked frequency of rickets in certain families, in parents and their offspring. However, quite apart from the question of the reliability of statistics embracing such a long period of time, it is evident that it is impossible by this simple method to exclude other caustive factors. In view of the definite importance of other influences, heredity must be regarded as playing a minor role. At most it would seem to confer a pre-disposition to the disorder—a field of genetics which remains as yet difficult of access.

It is probable, however, that there is a prenatal or etiologic factor, that the diet and regimen of the pregnant mother plays a role in the development of rickets in the infant. When we consider that the calcium is laid down in the fetus mainly during the last two months of intra-uterine life, and that premature infants are especially prone to the development of rickets, it is evident that infants may well be born with a constitution which, in one or more respects, predisposes to this disorder. The study of this question has recently been taken up experimentally by Korenshevsky,† in relation to the calcium and vitamin A content of the mother's diet. From a clinical point of view the well-known fact that twins have a special tendency to develop rickets, may be interpreted as being partly due to the subnormal quota of antirachitic constituents stored in their tissues. Furthermore, the variable susceptibility of infants to rickets—an observation first made by Glisson—probably is to be ascribed in part to prenatal differences in nutrition.

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* Siegert, F., Jahrb. f. Kinderh., lviii (1903), p. 929.

† Korenshefsky, V., Med. Research Committee, Special Report No. 71, London (1922).

The question of the occurrence of prenatal factors in rickets must be differentiated from that of fetal or congenital rickets. Whether congenital or intra-uterine rickets ever occurs cannot be definitely stated as the number of cases examined is too few to permit dogmatic generalization. It certainly must be extremely rare, for Schmorl was unable to observe rickets histologically in the bones of approximately 100 newborn infants; these cases included the ribs and long bones of many premature infants. Possibly rickets exists in utero in a form so larval and undeveloped that it has not given rise to focal and visible tissue changes, but has merely exerted a systemic nutritional alteration; a case of rickets in an infant of three months, which I recently met with, possibly may be thus interpreted.

The chief etiologic factors in rickets are not prenatal but postnatal. These influences may broadly be grouped under the captions of dietetic and hygienic. For years—indeed centuries—physicians have been divided into two camps in relation to this question, some contending that rickets is due entirely to defective diet, and others that defective hygiene is entirely at fault. Recently this question has been greatly clarified. Today we know that both factors play a part in its etiology, so that the discussion has resolved itself rather into an attempt to appraise their relative importance.

Let us first consider the question of diet. It is generally agreed that infants who have been nursed do not develop rickets as frequently as those who have been fed on cow's milk. This is well-founded clinical experience and in itself proves the importance of diet in the etiology. In my opinion, however, the protective value of human milk has been somewhat exaggerated. Some years ago in a study of negro infants, Hess and Unger found that in spite of breast feeding the great majority developed rickets, as judged by clinical examination. In a more recent investigation it was found that among the Russian Jewish population in the tenement district of New York City, 40 per cent. of breast fed infants showed radiologic signs of rickets (Table 1).*

There are no figures available as to the percentage of bottle-fed infants which develop rickets. If the newer criteria are employed—clinical, radiologic and chemical—certainly more than 50 per cent. of infants will gradually fall into this category by the end of the winter months.

The fact that bottle-fed infants are very prone to rickets proves that cow's milk can possess but slight antirachitic property. This does not depend on the purity of the milk, for rickets will develop even when the best (certified) milk is fed. Furthermore, feeding large quantities of milk furnishes no protection, indeed in some instances, it has seemed to lead to rickets. This fact must be emphasized because the textbooks contain statements to the contrary, and lead to the impression that if sufficient wholesome milk is fed, especially when given in proper dilution, rickets does not occur. Among the bottle-fed children it is generally considered that those receiving sweetened condensed milk, and milk prepared with proprietary cereal foods show the greatest percentage of rickets. This has not been my experience. In the course of careful observations I have not found that condensed milk led to rickets, nor a greater incidence of rickets among children who received milk diluted with various cereals, than among others who received milk diluted with water. There is indeed no established diet which can be particularly associated with the development of rickets; in this respect rickets differs from infantile scurvy. In my experience it has occurred very frequently on a diet of Finkelstein's protein milk. Many have associated a deficiency of fat with the occurrence of rickets, but in a series of infants who received milk in which the fat was replaced by vegetable oil, no such tendency was observed. On the other hand, rickets was noted clinically and by Roentgen-ray in infants given milk mixtures which were rich in fat. Moreover, animal experiments have shown that butter possesses but slight antirachitic power, and that it must be added to the dietary of rats in large percentage in order to bring about any protective effect whatsoever. It is difficult to interpret the general statements found throughout the pediatric literature to the effect that diets low in fat are most apt to lead to rickets. Empiricism has proved to be correct so often in regard to the relation of food and nutritional disturbances that due weight must be given to any conception which has existed for a long period of years. Although it is impossible to interpret this prevailing opinion, it must be remembered that until recently hygiene, and more particularly the effect of sunlight has been entirely left out of consideration. The rickets producing effect of carbohydrate would seem to be due largely to the fact that it leads to rapid growth. For a full discussion of this question the reader is referred to the recent monograph of Park.*

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* These examinations were carried out in March when rickets is most prevalent. No doubt, the percentage would have been far less had the survey been carried out some months earlier or later. The babies were a particularly well-nourished group, and the diet of the mothers was liberal, containing an abundance of animal fat. A similar group of infants showed only 20 per cent. of rickets radiologically in 1923, thus emphasizing the variable incidence of this disorder.

The importance of growth in the etiology of rickets has only recently been fully evaluated. For many years clinic observation taught us that very fat babies develop rickets whereas atrophic infants do not. However, the intimate relation of growth to rickets was not appreciated. It is now realized that, other etiologic factors being the same, the occurrence of rickets will depend largely on the rate of growth. On analysing the cases of rickets occurring in the infant asylum I found that about 75 per cent. developed among the well-nourished and but 25 per cent. among the poorly nourished infants. Premature infants owe their marked susceptibility to rickets largely to the intensity of their growth impulse. In this regard experiments

* Park, E. A., Physiological Reviews, iii (1923), p. 106.

on animals coincide with clinic experience so closely that tests are not considered comparable where growth has not taken place approximately at the same rate. The comparative significance of growth in weight and growth in length has not yet been determined.

The time is not yet ripe to discuss the question of the chemical nature of the deficiency or defect in the diet which leads to rickets. It is evident that it is not simply a deficiency of calcium or of phosphorus. If such were the case rickets would occur more often on a diet of human than of cow's milk, since the latter is much richer in both of these salts. Nor is the question one of the ratio of these two ingredients to each other, as the ratio remains unchanged whatsoever the degree of dilution of the milk. Recently Zucker* has suggested that the acid-base ratio of the food may play an important role, and that it may significantly alter the hydrogen ion content of the gastro-intestinal juices. It is, however, premature to do more than refer to this aspect of the subject.

The theory of the etiology of rickets which has been accorded greatest attention in the past few years is that it is due to a lack of vitamin A, the so-called fat-soluble vitamin, or of a vitamin closely associated with this factor. This hypothesis has been based purely on animal experiments, and almost entirely on the experiments with dogs carried out by Mellanby. There have been no clinic reports which would support this theory. Where a lack of this vitamin has existed in the diet of infants, the result, as reported by Block and others, has been xerophthalmia or keratomalacia and not rickets. The studies of Hess and Unger† showed that diets which were particularly poor in this vitamin did not lead to rickets, whereas in many instances diets which were rich in this factor, containing, for example, a large amount of milk, did not preclude the development of rickets. Studies on rats where this vitamin was excluded from the dietary lead to the development of characteristic eye trouble but not to rickets. So that it would appear that although this vitamin seems to have an influence in bone formation, it is not the specific antirachitic factor, and that, furthermore, the specificity of cod-liver oil in the prevention and cure of rickets is due not to its exceptionally large content of vitamin A but must be attributed to some other factor.

Probably the greatest recent advance in our knowledge of rickets has been the realization of the part which hygiene plays in its etiology. It is true that centuries ago Glisson believed that hygiene played a rôle in its causation. This aspect was also emphasized by Kassowitz,‡ who propounded the theory of a baneful influence of polluted air, and again by von Hansemann§ who brought forward the hypothesis of "domestication" as the main causative factor. But until recently

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* Zucker, T. F., Johnson, W. C., and Barnett, M., Proc. Soc. Exper. Biol., and Med., xx (1922), p. 20.

† Hess, A. F., and Unger, L. J., J. A. M. A., lxxiv (1920), p. 217.

‡ Kassowitz, M., "Gesammelte Abhandlungen, etc." Berlin (1914).

§ Hansemann, V. D., Berl. klin. Woch., xliii (1906), pp. 629, 670.

TABLE 1.—INCIDENCE OF RICKETS IN MARCH AMONG 25 BREAST FED INFANTS (BABY WELFARE STATION)

Case	Age, months	Weight, pounds	Condition	Head	Fontanelle, inches	Craniotabes	Teeth	Rosary	Epiphyses	Bow legs	Sits	Pot belly	Diastasis recti	Umbilical hernia	Muscles	Spleen palpable	Radiograph		Remarks
																	Epiphyses	Carpal centers	
1	6	13 14/16	Good	......	2 × 2	+	0	+	0	+	0	0	+	0	Good	0	Rickets	2	
2	9	21 2/16	Good	......	1 × 1	0	4	0	0	0	Sits	+	+	0	Good	0	Spreading	2	Receives cereal
3	6	14 12/16	Good	......	1¼ × 1¼	+	0	Slight	0	0	Sits	0	+	0	Good	0	Rickets	2	
4	4	12	Good	......	1½ × 1½	0	0	Slight	+	0	0	0	+	++	Soft	0	Rickets	1	
5	9	12 8/16	Pale	Square	1½ × 1¼	0	0	+	0	+	0	0	+	0	Fair	0	Rickets	2	
6	4½	12	Good	......	¾ × 1	0	0	Slight	0	0	0	0	0	0	Good	0	Negative	2	
7	10	15 8/16	Fair	......	¾ × ¾	0	0	+	+	+	0	+	++	+	Poor	4 cm.	Negative	3 and radius	Had measles; indoor three months; receives cereal and broth
8	6	18 8/16	Good	......	1¼ × 1	0	0	0	0	0	Sits	0	+	0	Good	0	Negative		
9	8	16 12/16	Poor	......	½ × ½	0	0	+	0	0	Sits	0	++	+++	Poor	0	Spreading	2	
10	6	19	Good	......	1½ × 1	0	0	Slight	0	0	0	0	+	0	Fair	2.5 cm.	Rickets	3	Outdoors once a week
11	8	15	Fair	......	2 × 2	+±	0	+±	+±	+	0	+	++	+	Poor	0	Rickets marked	1	Outdoors once a week receives milk with zwiebach
12	5	18	Fair	......	1½ × 1¼	+	0	Slight	0	0	0	0	+	0	Soft	0	Rickets	2	Outdoors every day
13	6	15 8/16	Good	......	1 × 1	0	0	Slight	0	0	Sits	..	++	0	Soft	2.5 cm.	Spreading	2	Never outdoors
14	9	20 8/16	Good	......	1½ × 1	0	0	0	0	+	Sits	+	++	0	Soft	4.0 cm.	Negative	2	
15	6	17 7/16	Good	......	1½ × 1	0	0	Slight	0	0	0	0	++	++	Poor	0	Negative	2	
16	4	15 6/16	Good	......	¾ × ¾	0	0	0	0	0	0	0	+	0	Good	0	Rickets(?)	2	Never outdoors
17	4	10 14/16	Fair	......	1 × 1¼	+	0	0	0	0	0	0	++	+	Poor	4.0 cm.	Negative	2	
18	5½	16	Good	......	1½ × 1½	+	0	+	0	+	0	0	++	0	Fair	0	Rickets	2	Out every day
19	4	11 11/16	Fair	......	2 × 2	0	0	0	0	0	0	0	++	0	Good	0	Negative	0	Out every day
20	5	16 4/16	Good	......	1¼ × 1	0	0	0	0	0	0	..	+	0	Good	0	Negative	0	Out every day
21	9	17 7/16	Fair	......	1½ × ¾	0	2	+	0	0	Sits	..	+	0	Good	0	Negative	2 and radius	Out every day; receive cereal
22	6½	16 11/16	Good	......	1½ × 1½	0	0	0	+	+	0	..	++	0	Fair	0	Negative	2	Out every day; receives cereal
23	5	16	Good	......	2 × 2	+	0	+	+	0	0	..	++	0	Good	0	Rickets moderate	2	Out once a week
24	7	16 9/16	Good	......	1 × ¾	0	0	+	0	0	0	..	++	0	Good	0	Rickets	2 and	Out every day
25	4	16	Good	......	1½ × 1½	0	0	0	0	0	0	0	++	0	Fair	2.0 cm.	Negative	2	Out every day
	Total	....	......	......	....	7+	..	9+	5+	..	..	..	..	..	..	...	10 rickets		

these hypotheses were insufficiently supported and, therefore, unconvincing. For some years Findlay* has stressed the importance of lack of exercise in the etiology of rickets, supporting his thesis by animal experiments as well as clinic investigations.

The subject, however, recently has passed from the realm of hypothesis to that of fact. It was shown by Huldschinsky† that rickets can be definitely cured, as noted by the radiograph, by means of frequent exposures to the rays of the mercury vapor quartz lamp. Hess and Unger‡ showed that a similar cure can be brought about, although the diet remains unchanged, when infants are exposed to the rays of the sun. These experiments showing the efficacy of artificial radiation and the sun's rays have been amplified and substantiated by experiments on rats. Rats fed standard rickets-producing diets can be regularly protected or cured by treatment of this kind. It is evident, therefore, that sunlight must play a rôle in the development of rickets. It is probable that we have here the chief explanation of the peculiar seasonal incidence of rickets, that its development in the winter and early spring, and its subsidence in the early summer, is to be accounted for mainly by the degree of ultra violet light which is afforded infants during these periods (Fig. 160). Other factors probably play a rôle, such as fresh air, exercise and cleanliness, etc. The fact, however, that artificial radiation is of value where all other hygienic factors remain unchanged, proves the great importance of certain light rays unassociated with fresh air.

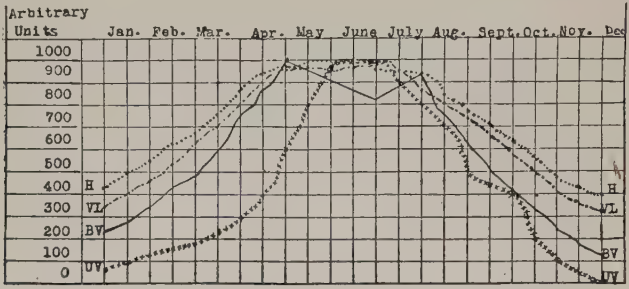

FIG. 160.—Seasonal variations of the sun's spectrum: *H*, solar heat; *V L*, visible light; *N V*, blue violet; *U V*, ultraviolet.

This aspect is so new and so difficult that it is impossible at the present time, to appraise the respective values of the dietary and the hygienic factors in the etiology of rickets. Moreover, its importance must vary according to attendant circumstances. It is clear that where the diet is largely at fault, this will lead to a large proportion of rickets, whereas in large metropoles where the atmosphere is murky, the weather damp and sunless, the housing conditions crowded, hygiene will play a more dominant rôle. The fact that breast fed babies develop rickets so frequently in winter must be associated with the lack of sunlight. Furthermore, the excessive incidence of rickets among the Italian infants in the large cities of the United States, despite the fact that the majority are nursed, is attributable to the racial habit of keeping them indoors during the cold months. The marked susceptibility of negro infants to rickets in the northern cities, such as New York and Chicago, is to be attributed largely to a deficiency of sunlight. In the West Indies, the home of most of these negroes, rickets is stated, on good authority, to be almost a negligible disorder. In New York City fully two-thirds of the breast fed negro infants and almost all of the bottle-fed infants show signs of marked rickets. This is partly due to the fact that the pigment of their skins prevents the penetration of the protective rays of the sun, so that from an etiologic point of view, they are living in increased darkness. This phenomenon has been illustrated recently by an experiment which showed that the same degree of light which sufficed to protect white rats fed a standard diet, failed to protect the melanotic rats from rickets under similar conditions.*

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* Findlay, L., Lancet, i (1922), p. 825.

† Huldschinsky, K., Zeitschr. f. orthop. Chir., xxxix (1920), p. 426.

‡ Hess, A. F., and Unger, L. J., J. A. M. A., lxxvii (1921), p. 39.

Some are of the opinion that rickets is an infectious disease, due to bacteria or their toxins entering the circulation through the alimentary tract. In this respect the situation is similar to that which exists in regard to the etiology of scurvy, beriberi and pellagra. There is no convincing experimental evidence to support this view. About a generation ago Morpurgo† undertook a series of investigations in which he attempted to prove that rickets could be induced in rats by means of bacterial infection, by inoculations of a diplococcus. Although the lesions which he described were undoubtedly rachitic, the experiments cannot be interpreted as the diet was uncontrolled. Koch's‡ inoculations of the streptococcus longus into dogs are open to similar criticism. It cannot be gainsaid that infection may play a rôle in the etiology of rickets, but when we consider the clear cut experimental data which have been obtained by a simple alteration of the diet, or by means of ultra violet light, it would seem that this rôle must be of secondary importance. It is quite possible that infectious processes may at times disturb the normal metabolism. There is, however, no clinic or experimental evidence on which to base this assumption.

In this connection the relation of syphilitic infection to the development of rickets must be considered. As is well known, French clinicians for some generations have believed that a large proportion of rickets is syphilitic in origin. Today Marfan§ is the chief exponent of this opinion. In this country such is undoubtedly not the case; rickets is prevalent among infants who do not manifest any signs whatsoever of congenital syphilis. It is possible that syphilis may lead more readily to the development of rickets, especially in cases where the bones are involved. In an examination in the month of March of some syphilitic infants of rachitic age, I have failed to find evidences of rickets either clinically, radiologically or by a chemical examination of the blood.

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* Hess, A. F., Lancet, ii (1922), p. 367.

† Morpurgo, B., Verhandl. deutsch. path. Ges., iii (1901), p. 40.

‡ Koch, J., Zeitschr. f. Hyg. ŭ Infektionskrankh., lxix (1911), p. 436.

§ Marfan, P., Maladies des Os., Paris, Bailliere et Fils (1912).

The etiology of rickets has been attributed to a derangement of the glands of internal secretion, the endocrine glands. This has been based largely upon the experiments of Erdheim* who demonstrated the occurrence of rachitic lesions in rats following the extirpation of the parathyroid glands. These carefully conducted experiments are open to the criticism which applies to all the early investigations, namely, that the diets of the animals were uncontrolled. The fact that the parathyroid gland is associated with calcium metabolism as well as with the development of tetany, and that both of these conditions are related to rickets, has served to strengthen the opinion of those adhering to this theory. Furthermore, reports of the successful therapeutic use of extracts of endocrine glands have been brought forward to support this view. The thymus has also been held responsible. At the present time it is fruitless to discuss hypotheses resting on such insecure foundations; both the clinic and experimental evidence are too slim to be accepted. This does not imply, however, that the theory is by any means unreasonable, or that further experiments may not show that these glands play a rôle in the pathogenesis of rickets.

Metabolism.—Ever since metabolism studies have been carried out on infants, cases of rickets have constituted a favorite theme for study. The literature on this subject is voluminous but full of contradictions and has not furnished the results which might be expected. This is due to many causes. In the first place rickets is a disease of slow and gradual development, and metabolic tests of short duration may readily prove fallacious; furthermore, it is characterized by an irregular course, marked by frequent remissions, so that a test may include a period of inactivity and be misleading. The data of these studies is, moreover, so contradictory that it is evident that errors of technique have occurred, a complication which is not surprising when one considers the difficulties in carrying out successful metabolism investigations on young infants. There are numerous factors which tend to complicate studies of this kind in infants, from which similar studies on adults are comparatively free. Among these may be mentioned the frequency and importance of minor infections, and of transient periods of lack of growth, and the marked effect of ultraviolet light on the salt metabolism. In view of these circumstances, and the probability that, in the near future, there will be a thorough revision of this aspect of the subject, it seems advisable to treat metabolism from a broad rather than a detailed point of view.

Until recently attention has been focused mainly on calcium, other salts being accorded merely passing attention. The studies of Schabad,[*] however, have been an exception to this rule. Almost alone he appreciated the significance of phosphorus in rickets, definitely stating that it played even a greater rôle than calcium. More recently, as we shall see, Telfer[†] has come to the same conclusion, finding that the ratio of phosphorus retention is less than that of calcium.

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* Erdheim, J., "Rachitis ŭ Epithelkoerperchen," Vienna (1914).

Investigators have approached the subject by various paths; the ash and more particularly the calcium content of the body has been determined, the body exchanges of various salts in the diet have been compared to those of the urine and feces, and more recently, analyses of the blood have been carried out. The bodies of newborn and young infants have been analyzed in order to furnish normal data for estimation of the daily need of calcium. As the result of a careful consideration of a number of studies of this kind, Aron[‡] concluded that the body contained about 12 gm. of CaO per kg. body-weight and that about 1.2 gm. are needed per 100 gm. of increase in weight. The figures of Schabad were about the same (1.25 gm.) and led to the conclusion that an infant gaining 20 gm. a day, requires 0.25 gm. of calcium oxide. This method of computation is open to the criticism that bodies differ considerably in their ash content, and that some contain more fat than others. Orgler[§] gives 0.174 gm. as the amount required per 100 gm. of growth. Incidentally attention may be called to the great importance accorded to growth in regard to the calcium requirement, an importance not sufficiently emphasized in clinic studies.

TABLE 2.—ANALYSES OF BODIES OF NEWBORN INFANTS (ARON)

Author	Percentage of ash in body	Percentage of CaO in ash	Amount of CaO per kg. body weight grams
Hugouneng (1899)	3.55	40.48	14.39
Michel (1899)	3.37	41.39	13.97
Giacosa (1894)	3.35	41.92	13.72
Steinitz (1904)	3.04	37.90	11.52
De Lange (1900)	2.95	38.89	11.51
Soldner (1903)	2.66	38.08	10.12
Average	3.15	39.79	12.53

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[*] Schabad, I., Ztschr. f. klin. Med., lxvii (1909), p. 454; lxviii, p. 94; lxix (1910), p. 435.

[†] Telfer, S. V., Quart. J. Med., xvi (1922), pp. 45 and 63.

[‡] Aron, H., Biochem. Ztschr., viii (1908), p. 1.

[§] Orgler, A., Ergebn. d. inn. Med. u. Kinderh., viii (1912), p. 170.

Holt, Courtney and Fales* conclude that the minimum daily physiologic requirement is 0.13 gm. CaO per kg. body-weight for infants on a milk diet, and 0.09 gm. when on a mixed diet.

There is a general agreement that the bones of rachitic infants and animals are poor in calcium. Schabad demonstrated this some years ago for infants, and it has been corroborated by many others. The bones of rachitic pups and rats likewise have an abnormally low calcium content. The ratio of calcium and of phosphorus is, however, normal, remaining unaffected even in the most severe instances of the disorder. In the femora of infants, rachitic as well as normal, this ratio has been found to be 1.35 to 1 (CaO to P_2O_5). It is evident that this lack of calcium may be accounted for theoretically either as the result of decalcification of formed bone, or as a failure of calcification of new bone. At present the former explanation, that of halisteresis, is not generally accepted; the arguments for and against this hypothesis will be found in a recent paper of Findlay, Paton and Sharpe,† which contains an excellent summary and criticism of the literature to 1921 on the metabolism of calcium, as well as the authors' original investigations.‡ The majority of investigators believe that the deficiency of calcium is due to defective ossification, associated either with a deficient absorption from the digestive tract, or a defective utilization of normally absorbed calcium.

As might be expected, attention has been particularly directed to the composition of woman's milk, especially in cases where rickets has developed on this diet. The results of numerous studies of this kind are so divergent that the question must be considered as undecided. Some are of the opinion that the milk of mothers of rachitic infants is definitely deficient in calcium and in phosphorus, whereas others have published analyses which are within the field of the normal. It is evident that some of the tests have been carried out with insufficient quantities of milk, or have not been repeated for many daily periods. More work in this field is highly desirable. From a clinic point of view, in order to determine whether the amount of calcium was sufficient, cognizance will have to be taken of the requirement of the individual infant, as judged by its rate of growth. In view of the fact that rickets occurs much more frequently on a diet of cow's milk, even when fed in large quantities, it is impossible to believe that there can be a simple relationship between rickets and the amount of calcium or of other salts in the milk. This peculiar incidence points rather to other deficiencies, or to a faulty correlation of the constituents in cow's milk.

* Holt, L. E., Courtney, A. and Fales, H., Am. J. Dis. Child., xix (1920), p. 97.

† Findlay, L., Paton, N. and Sharpe, Quart. J. Med., xiv (1921), p. 352.

‡ A few years ago Hess and Unger carried out an investigation of "the diet of the negro mother in New York City." The dietaries of these women were found lacking chiefly in fresh vegetables and fruits; the calcium content was also insufficient (Jour. Amer. Med. Assoc., lxx (1918), p. 900).

Attention has been directed particularly to the excretion of calcium and phosphorus, and the metabolism of these salts after their absorption. Both of these aspects are still the subject of controversy. They have been so well summarized recently by Telfer* on the basis of his own work and that of his predecessors in this field, that we can do no better than quote his conclusions:

"The excretion of calcium, phosphorus, and fatty derivatives are independent, calcium being eliminated chiefly as phosphate and to a lesser extent as insoluble soaps, the relative amounts of these varying with the conditions of the intestinal contents. Twenty to thirty per cent. of the total CaO excreted normally exists in the form of calcium soaps. A large proportion of the ingested calcium and phosphorus is restricted to the gut, and appears in the feces as tri-calcium phosphate. Consequently the absorption of a considerable part of the intake of these is normally prevented. The total amount of calcium and phosphorus eliminated is nearly proportional to the intake. No evidence was obtained that mineral matter from endogenous sources was excreted in the feces to any appreciable extent.

"The urinary calcium is a very small fraction of the total calcium excreted. It is slightly increased by an increased intake of calcium salts, by the administration of acids salts, and, generally, by acid formation in the intestine. In normal infants 40 per cent. of the total phosphorus excreted appeared in the urine, 60 per cent. in the feces. With acid formation in the intestine, less phosphorus is excreted by the feces, more by the urine. It was found that the degree of deviation of phosphorus to the urine was roughly proportional to the amount of fatty acids in the feces, and, consequently to the extent to which fatty acids had displaced phosphoric acid from its normal combination with calcium in the intestine. It is argued that the phosphorus which is excreted by the feces has been in greater part restricted to the intestine owing to the presence of calcium salts, and cannot be regarded as a re-excretion into the intestine. The excess of phosphorus over the equivalent amount of calcium forming tri-calcium phosphate is absorbed, and a part of this is retained, the remainder being excreted in the urine. An excess of calcium salts restricts an increased quantity of phosphorus to the intestine. In a particular case, in which the intakes of fat and phosphorus were low, an excess of calcium in the diet rendered the urine phosphorus free.

With a persistent excess of fatty acids in the intestine a much greater proportion of calcium is excreted as soaps. In certain pathologic cases, in which an excessive soap formation was a feature, it is suggested that the common causative factor was a persistence of free fatty acids in the intestine, possibly secondary to some degree of hepatic insufficiency."

Telfer found an excess of P_2O_5 retained over the equivalent amount of CaO required for bone formation. It is of interest to note that in cases of rickets there was not only a diminished retention of calcium and phosphorus, but that the defect was more pronounced in the latter respect. This he attributes not to a lessened absorption of phosphorus, but to a diminished fixation by the calcium, the absorption of the latter being defective possibly as the result of some alteration in the gastro-intestinal functions. It appeared as if the fixation of the phosphorus was determined by the amount of calcium retained.

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* Telfer, S. V., Quart. J. Med., xvi (1922), pp. 54 and 63.

It should be remembered that rickets probably occurs most often in a latent form, as does infantile scurvy, a form which it is difficult to diagnose even by means of metabolism tests. During this period a negative balance of calcium and phosphorus may exist, or, quite possibly, the retention may merely be insufficient to meet the needs of a rapidly growing infant, and thus slowly lead to rickets after a period of many months—a subnormal but not a negative balance.

Schabad showed that cod-liver oil, when given in rickets, is able to change a negative to a positive calcium balance. In his first studies he combined this oil with phosphorus to which he ascribed the beneficent effect. Later, however, he realized that the retention was due to the oil. The following test, published in 1910, well illustrates this point:

	PERCENTAGE OF INGESTED CaO RETAINED
No treatment	4.79
Phosphorus + 0.1 olive oil	10.11
Sesame oil (2 teaspoonfuls)	0.97
Cod-liver oil (2 teaspoonfuls)	43.35
Cod-liver oil + phosphorus (2 teaspoonfuls)	72.49

In rickets when the blood is deficient in inorganic phosphate, cod-liver oil will regularly raise its content to the normal level. In tetany, where the calcium percentage is low, it will likewise remedy the deficiency, an effect which cannot be brought about by the administration of calcium salts.

Within the past few years we have gained a somewhat clearer understanding of the chemical changes which take place in the blood in the course of rickets. It is probable that within the near future this field of our knowledge will be greatly extended. For some time it has been known that the blood of rachitic children contains a normal percentage of calcium, or a percentage but slightly below the normal. In view of the well-established negative calcium balance, it was suggested that although the total calcium content was unaltered, the form in which the calcium occurred might be less active. Experiments by Rona and Takahashi* on animals, as well as tests carried out recently by Meysenbug and McCann† on infants have shown that the amount of diffusible calcium is normal in amount. The latter conclude that

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* Rona, P., Takahashi, D., Biochem. Ztschr., xxxi (1911), p. 336.

† Meysenbug, L., McCann, G., J. Biol. Chem., xxxxvii (1921), pp. 529–541.

"the diffusible calcium of the serum in experimental tetany in the dog and in human rickets is between 60 and 70 per cent. of the total serum calcium.

Iversen and Lenstrup* as well as Howland and Kramer† have recently demonstrated that the inorganic phosphorus of the blood is lower than normal in rickets. In moderate cases there is a decrease of 20 or 25 per cent. and in some cases this may amount to over 50 per cent. The interpretation of this phenomenon in its relation to the bone lesions is not yet understood. Clinically rachitic changes may be noted before the blood phosphate is appreciably diminished. In rats although lowered inorganic phosphate generally accompanies rickets, this may be encountered where rickets does not exist, or again, rickets may undergo cure in spite of the fact that the phosphate level remains well within the rachitic zone. It is too early to attempt to interpret these new observations, especially as we know nothing about the function of other forms of phosphorus in the blood, for example: The so-called "acid soluble" and the organic phosphorus.

In view of the fact that a lack of calcium in the food could not account for the development of rickets and that the percentage in the blood was found normal, it is but natural that the deficient calcification in the bones has been attributed to a defect in the bone itself. This theory is not new, but has formed the basis recently of several series of experiments. In the main, these have consisted of comparing the adsorbtive capacity of the bones of rachitic and non-rachitic infants for calcium in vitro, or within the animal body. It may be stated in general that these experiments have been unconvincing. As the result of experiments of this nature, Pfaundler has suggested that there is a lack of specific affinity in the rachitic bones. Others, for example: Von Recklinghausen and Ribbert, have predicated a toxic factor in the blood which causes injury to the osseous and cartilaginous tissue throughout the body. Marfan believes that the bone marrow is at fault in this regard.

From this brief survey of the studies of metabolism in rickets it will be evident that we are still in ignorance of the basic principles underlying its pathogenesis. We have no knowledge whatsoever of the intermediary metabolism occurring in the course of the disorder. Possibly this information will be forthcoming as the result of experiments on animals, but it is more probable that it will be gained from investigations on human rickets. Although the anatomical lesions of rickets in rats and in human beings appear identical, the physiologic mechanisms in the two species differ sufficiently to render dangerous a simple transference of metabolic data from one to the other.

As the result of the realization of the important rôle which light plays in the development and cure of rickets, a new field of metabolic investigation must be developed in relation to this disorder. Little is known in regard to the metabolism of light in general, and nothing of its action in rickets. Hess and Gutman* showed that in the course of treating cases of rickets with sunlight, the lowered percentage of inorganic phosphate in the blood steadily approached the normal level. This phenomenon can be brought about also in the course of curing rickets by means of the radiations from the mercury vapor quartz lamp or the carbon arc lamp. In this connection reference may be made to the† "seasonal tide" of blood phosphate, a tide corresponding to the curve of the ultra violet radiations of the sun. This manifestation in bottle-fed babies of a "phosphate tide" is undoubtedly linked with rickets, and may well be associated with other chemical changes of a seasonal nature.

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* Iversen, P., Lenstrup, L., Forhandl. v. Förste Nordiske Kongres f. Paediatri (1920). (Cited by Howland and Kramer.)

† Howland, J., Kramer, B., Am. J. Dis. Child., xxii (1921), p. 105.

Symptomatology.—Rickets is probably more often overlooked than any other of the common disorders of childhood; the number of cases which pass unrecognized is greater than that which is diagnosed. This is due partly to the inherent difficulties of early diagnosis, but largely because rickets is not specifically looked for. Until recently, it has suffered the consequence of being for teacher, as well as for student, a most uninteresting disorder. As is true of infantile scurvy and other abnormal nutritional states, rickets is present generally in a form so incompletely developed that it does not attract attention. The earliest symptoms are restlessness, irritability and head sweating, of which the latter is the most significant. But even head sweating is a sign which is suggestive rather than indicative, and must await further corroboration. The sweating generally is confined to the head, and may lead to loss of hair over the occipital region, especially when it is accompanied by head rolling. The typical early case of rickets is that of a baby in the second half year of life, fairly well nourished and normal to casual examination, but which detailed clinic investigation demonstrates to depart from the norm in many respects. Its head may be somewhat square in shape, and the fontanelle too widely open, with borders thin and yielding too readily to pressure. Areas of softness may be found on the lower aspect of the occipital bones or lateral portions of the parietal bones. Teething is delayed, as well as the ability to sit or to stand. Moderate enlargement of the costochondral junctions will be found if sought for. The musculature is poorly developed, but not atrophic or wasted. Radiographs of the epiphyses at the wrist may show changes in the ulna typical of rickets. A test of the blood will show a deficient percentage of inorganic phosphate. Naturally all of these signs or symptoms will not be present in every case. In an infant which seems to be thriving, and who is of average weight and healthy in appearance, such slight divergencies from the normal may well be overlooked unless a routine search is made.

The well-developed or advanced case presents a picture which is apparent at a glance and requires but little experience to recognize

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* Hess, A. F., Gutman, M., J. A. M. A., lxxviii (1922), p. 29.

† Hess, A. F., and Lundagen, M. A., J. A. M. A., lxxix (1922), p. 2210.

(Figs. 161, 162). The head has become square and perhaps slightly enlarged. The thorax is deformed and shows not only two lateral rows of visibly enlarged costochondral junctions, but also a groove traversing its lower part, or deep furrows compressing and deforming it on either side. The abdomen is markedly large and protruberant and constitutes a striking feature of the infant, which at this stage is generally more or less poorly nourished. The legs are bowed, and the posture of the infant may give evidence of laxity of the ligaments. Roentgenologic examination shows advanced involvement of the lower epiphyses of the ulna and of the radius, and the blood phosphorus is greatly reduced.

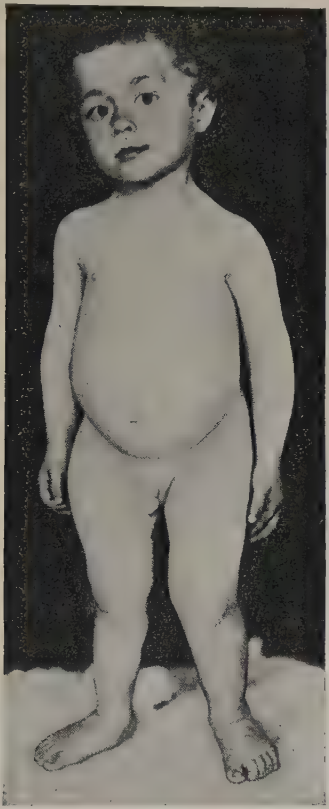

Fig. 161.—Typical rachitic child. Large head. Pot belly. Knock-knees. Flat foot.

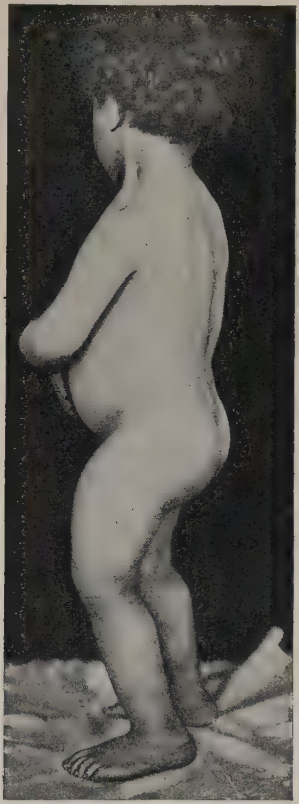

Fig. 162.—Typical rachitic child. Note posture and anterior bowing of thighs.

The changes in the *cranial bones* have always attracted particular attention in connection with rickets. This is due no doubt to the fact that they form such a striking sign in the classical case. The prominent frontal and parietal bosses which are so evident in advanced cases, and which are especially developed in negro infants, are present only in rickets and in congenital syphilis. These protruberances may be so marked as to constitute a "caput quadratum," the square head of rickets. At times longitudinal and transverse grooves divide the calvarium into four distinct areas, producing the so-called "hot cross bun" appearance. The anterior fontanelle is generally enlarged, and, as just mentioned, its borders are thin and do not present the normal resistance to pressure.

Craniotabes is regarded by many as being the earliest and most reliable sign of rickets. There can be no doubt of its diagnostic value, but it cannot be regarded by any means as pathognomonic. Its absence possesses no diagnostic significance whatsoever; its presence must be considered with various reservations. In the first place, craniotabes must be differentiated from the softening of the occipital or parietal bones described by Elsasser many years ago, and which is not a sign of rickets as commonly defined today. This softening is more or less extensive, generally situated along the suture lines, and is encountered in typical form in premature and in very young infants. These babies may later present no evidences of rickets. The osteoporosis heals under treatment with calcium salts. The true craniotabes is the small soft compressible area, or areas, in the lower part of the occipital or parietal bones. These areas have a marked tendency to heal and calcify, so that although this sign could be elicited in 30 per cent. of the babies in the infant asylum at three months of age, it was present in only 10 per cent. at six months of age. It possesses, therefore, its greatest diagnostic significance after the fifth or sixth month, when the congenital softening (if not too extensive) has healed. Craniotabes frequently disappears during and despite the development of the characteristic clinic, radiologic and chemical signs. In babies under six months of age it has been associated in numerous instances with a normal inorganic phosphate content of the blood. The question of the significance of craniotabes is closely linked with that of the relationship of osteoporosis and rickets, a subject which is still debated by pathologists, clinicians and biologists.

A great deal of stress in relation to the diagnosis of rickets is placed on the delayed eruption of the deciduous *teeth*. This sign is most untrustworthy. No doubt rachitic infants in general teethe later and more irregularly than the non-rachitic, a phenomenon which is especially marked in severe cases. In the mild cases, however, which constitute those commonly met with, this sign is fallacious. Family idiosyncrasy, mental backwardness and other causes will lead to delayed dentition quite in the absence of rickets.

A subject of great social importance is that of the influence of rickets on the formation and on caries of the deciduous and the permanent teeth. This question is not new but has been brought to the foreground once more by the renewed interest in rickets and the interesting experimental studies of May Mellanby* and others, on the relation of dental caries to the diet. Dick,* who has studied this aspect of the subject most carefully, believes that "the occurrence of rickets in early infancy is the most potent factor in the production of defective teeth." He refers especially to the permanent teeth and describes a condition where there is a defect of the enamel of the incisors, more especially of the central incisors—enamel which has been laid down during the first two years of life—as being pathognomonic of infantile rickets. The temporary teeth do not seem to be greatly affected in the cases which I have observed, probably due to the fact that for the most part they are calcified before birth or during the first months of life. It is impossible to present the subject of the interrelationship of rickets and dental caries in statistical form, as one cannot be certain that other etiologic factors have been excluded. For example: In order to be certain that rickets is responsible for the dental caries and hypoplasia of school children, one should know that the diet has contained sufficient inorganic salts and vitamins, and that local or systemic infection has not played a rôle. In severe cases not only the teeth but the shape of the jaw may be affected, leading to a protrusion of the anterior part of the alveolar arch, which results in deformity and malocclusion of the teeth. A frequent deformity consists in lateral constructions of the lower jaw at the site of the canine teeth.

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* Mellanby, May, The Lancet, ii (Dec. 7, 1919), p. 769.

The *thorax* is almost always involved in the course of rickets. The typical change is the enlargement of the costochondral junctions known as "beading" of the ribs and leading to the formation of a "rachitic rosary." In its marked and fully developed form, the rosary is evidenced by a double row of circumscribed knobs or elevations running almost longitudinally on either side of the chest wall. It is distinctive of rickets, and is capable of being confused only with the "scorbutic rosary," an aspect which will be discussed in considering diagnosis. In two instances I have found it present in marked degree in diabetic infants. The difficulty in regard to this sign lies in defining the term rachitic beading and in determining what should be considered a degree of enlargement compatible with normal development. The criterion—like many other clinic evaluations—can be learned only by actual experience. A slight degree of beading should not be too greatly stressed, especially the minimal degree which is so frequently encountered in the newborn or in infants during the first few months of life. The common error, however, is rather a failure to examine carefully for beading, or to palpate along a line internal to the costochondral junctions. The enlargement generally involves the middle tier of the ribs, the fifth, sixth, seventh and eighth, and may be difficult to elicit if the ribs are overlaid with fat. In an experience of about 2000 physical examinations carried out in the course of a study of rickets, beading of the ribs was found to be the most constant early clinic sign *e.g.*, Table 3, and greater reliance has been placed on it than on craniotabes,

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* Dick, L. J., Brit. J. Child. Dis. (1916), p. 332.

TABLE 3.—FIRST SIGN OF RICKETS (51 CASES)

	Dried milk	Raw milk	Protein milk	Total
Rosary	14	9	1	24
Roentgen-ray	1	0	1	2
Blood phosphorus	4	2	1	7
Rosary + Roentgen-ray	1	0	0	1
Rosary + phosphorus	5	2	1	8
Roentgen-ray + phosphorus	2	0	0	2
Rosary + Roentgen-ray + phosphorus	2	3	2	7
Total	29	16	6	51

bowing of the legs, head sweating or even the radiographic films. Its especial value is as an early sign at the middle of the first year of life. In the course of the second year it frequently becomes obliterated by the recession of the costochondral junctions, which accompanies the formation of the well-known "Harrison's groove." This constitutes a depression, or gentle concavity, running transversely across the lower part of the chest, at the level of the attachment of the diaphragm. A sharp distinction should be made between the value of beading of the ribs for early diagnosis and as a criterion of healing. No reliance can be placed on this sign in judging of the value of curative agents, nor does the presence of a "rosary" indicate that an infant has active rickets.

Mention has been made of the horizontal groove on the lower chest wall termed Harrison's groove. This deformity is frequently met with in even mild cases but generally disappears in later childhood or is indicated merely by an outward flanging of the lower borders of the ribs. In severer cases longitudinal depressions may occur either anterior or posterior to the rosary. These may reach such a degree, as has recently been emphasized by Park* and his associates, that pressure is exerted on the lungs, leading to disturbances of respiration. In marked cases, such as are seen most often in colored infants, there may be a projection of the sternum with lateral depressions on either side, or a sinking of the sternum, resulting from weakening at the costochondral junctions. In the former instance, we have the typical "chicken breast," in the latter the "funnel-shaped chest." Either of these deformities may come about, however, from other causes, and is not diagnostic of a previous rachitic process. Although the typical rachitic thorax is abnormally large in the anterior posterior diameter, not infrequently the chest is flattened and enlarged in the horizontal diameter. Associated with this condition we often find an undue softening or compressibility of the sternal region, an abnormal mobility of the sternum on the costal cartilages. In my experience this condition has been frequently linked with subsequent development of bowing of the legs and has come about on a diet of sweetened condensed milk.

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* Park, E. A., Shipley, P. G., Am. J. Dis. Child., xxiii (1922), p. 91.

Spinal curvature is another abnormality brought about by rickets. This deformity is of little value in early diagnosis, as it is difficult to interpret mild degrees of kyphosis in young infants who either cannot sit, or sit with difficulty. It may, however, constitute an important residual sign of rickets. Kyphosis is the common form of curvature and differs from tuberculous kyphosis in being less angular, not tender and readily reducible on traction. These differential criteria suffice for the great majority of cases, but occasionally the diagnosis presents difficulties. In a case of this character there was definite local tenderness of the lower dorsal spine, although no disorder other than rickets was discoverable, either at the time, or for months thereafter.

This question leads to a consideration of the occurrence of pain and tenderness in rickets. Descriptions of rickets of 20 or 30 years ago emphasized tenderness as one of the important signs. Even Glisson, who was the first to differentiate between rickets and scurvy, mentions pain and tenderness among the symptoms. In mild rickets I have never encountered tenderness of the bones, or of the soft parts. In some cases of severe rickets tenderness has seemed to exist, although these infants have been in such a miserable condition, that the whining or crying which followed pressure of the thorax or of the long bones, was difficult to interpret. Undoubtedly, most of the tenderness which has been attributed to rickets has been due to scurvy; when tenderness is encountered in rickets careful inquiry should be made regarding the antiscorbutic adequacy of the diet.

Almost any of the bones may be involved in the rachitic process, provided the disorder is sufficiently advanced. In the most intense cases where there is rachitic dwarfism, we find deformity of the entire skeleton and few bones seem to be normal. The long bones of the upper extremity are less often affected than those of the lower extremity, except in regard to *enlargement of the epiphyses.* This classical sign involves most often the lower end of the radius, next in frequency the lower end of the tibia, and somewhat less often the head of the tibia and abutting epiphyses of the femur. Viewed from the standpoint of early diagnosis, enlargement of the epiphyses is of secondary importance, for it is very difficult to determine slight swelling of the bones, so that by the time the enlargement is definite the diagnosis has been established from the beading of the ribs or other signs. As would be expected, rickets can be diagnosed by radiographs of the wrist before the epiphyses are definitely enlarged to palpation. Such is the rule. In several instances, however, this rule did not hold—there was definite enlargement at the wrist although none of the typical changes were evident by Roentgen-ray.

It is surprising that *radiography* was not employed sooner in the diagnosis of rickets, especially when one considers that its value was unduly stressed in relation to infantile scurvy. Two monographs were published in Germany on this subject as early as 1911,*† but they passed unnoticed. The characteristic early change occurs at the lower epiphysis of the ulna. This becomes flattened or broadened, its inferior surface is no longer sharp and straight, but concave and irregular. This surface may appear irregular and as if covered by an uneven fringe (Fig. 163). The radius is similarly altered as the disease progresses. The shafts of the bones appear reticular and less well calcified, and the structure is evident in too great detail. The periosteum occasionally gives the appearance of unevenness and irregularity. Development of the carpal centres may be delayed.

Fig. 163.—Marked rickets. Concavity and fraying of epiphyses. Abnormal visibility of bony structure.

It may seem illogical that beading of the ribs can be noted by palpation before changes are apparent by Roentgen-ray. It must be borne in mind, however, that could we obtain satisfactory radiographs of the costochondral junctions, diagnosis might be first established by Roentgen-ray. Moreover, changes undoubtedly occur in the epiphyses of the wrist before we can definitely distinguish them; that such is the case is frequently shown by the experience that specific therapy leads to visible calcification in epiphyses which appeared normal by Roentgen-ray. In judging of the healing of rickets, especially of the value of therapeutic agents, the radiograph is invaluable and frequently gives evidence before there is a definite rise in the inorganic phosphate of the blood. The film shows an irregular line of calcification, which soon becomes a band; later it is noted that a new tip has been added to the end of the bone. The shafts also become denser. The carpal centers ossify, or existing centers become circumscribed by a ring of inorganic salts. Finally, the epiphyses become healed, the concavities are filled in, but for many months the "scars" are visible, which inform us that rickets has been present—an abnormal contour or bowing may be noted for years.

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* Fraenkel, E., and Lorey, A., Archiv. u. Atlas d. Normalen u Patholog., Anatomie in typisch. Roentgenbild, Hamburg, Graef und Sollem (1910).

† Wohlauer, F., Atlas u Grundriss d. Rachitis, Muenchen, J. F. Lehmann (1911).

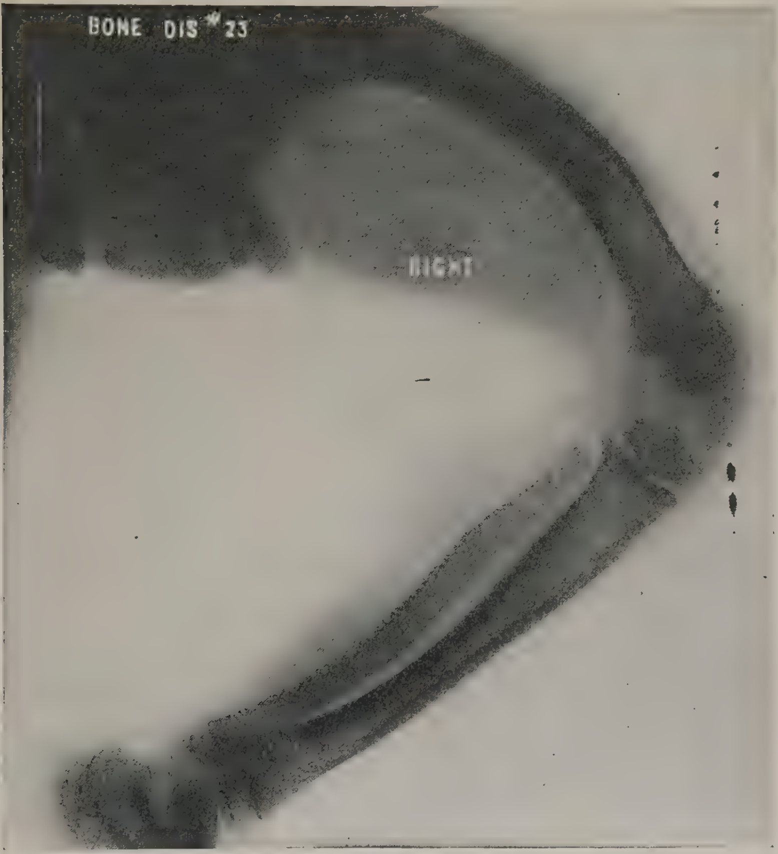

Fig. 164.—Residual rickets. Bowing of femur, fibula, and tibia.

As stated, rickets does not involve the upper extremities to the same degree as the lower extremities. The humerus is generally spared excepting in severe cases. The clavicle is frequently bent, and not infrequently, the site of fracture at the junction of its inner third and outer two-thirds; this may lead to permanent thickening at this site. The scapula may show osteoporosis or deformity. Fractures of the bones of the forearm or of the humerus are not infrequent and may give no sign or symptom whatsoever, being discovered accidentally in the course of routine roentgenologic examination.

As is well known, the lower extremities frequently are involved in the rachitic process (Figs. 164, 165). The shaft of the femur bows forward, its neck becomes depressed and horizontal, bringing about the pathologic and clinic condition known as coxa vara. The common lesion of the tibiæ is a bending inward of the lower third or fourth of the shaft, producing "bow legs." This may be of mild degree, or so severe as to require surgical intervention. It is surprising, however, to what extent nature tends to straighten these curvatures. There is no doubt but that the weight of the body, especially in heavy babies who walk early, aids in producing bowing; however, the most marked examples of this lesion are frequently seen in infants who have never walked. Attention should be called to the bowing of the legs of newborn and very young infants, a condition quite unassociated with rickets. In addition to this lateral bowing of the tibia there is a rachitic backward bending of the lower end of the shaft. This develops only in very severe cases and shows far less tendency to spontaneous correction than the other form. Instead of bow legs, a condition of knock-knee may develop, or even a combination of both. Associated with these deformities there is generally a marked laxity of the ligaments of the knee joint. When the ligaments of the bones of the foot are involved the plantar arch may sink and flat-foot develop. Rickets, however, is not the sole cause of flat-foot.

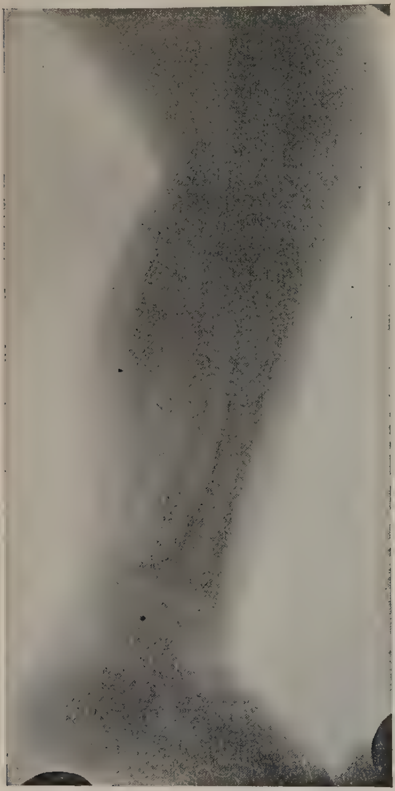
FIG. 165.—Same case as Fig. 163. Typical marked rachitic lesions of epiphyses of upper and lower ends of tibia.

The *general nutrition* usually does not suffer in rickets, provided that the appetite is good and digestion remains undisturbed. One of the most surprising features is the unreliability of the general appearance in the early diagnosis of rickets. In the infant asylum, investigation of this aspect disclosed the fact that three-quarters of the babies developing rickets had thrived well. In fact, it is the infant making abnormally large gains which is most apt to show signs of rickets in the course of the winter. This is one reason for the predisposition of premature babies to rickets, for, as is well known, their rate of growth is more rapid than that of the full term infant. The relation of growth to rickets has only recently been appreciated and is largely due to observations on experimental animals. Puppies and rats which grow rapidly have a marked tendency to develop rickets, whereas those which gain but little require far more stringent diet to bring about the disorder. Such observations furnish the explanation of the failure of atrophic infants to develop rickets, and of spontaneous cure following diarrhea or a low food intake. They likewise explain in part the tendency to spontaneous cure during the second year of life when the growth impetus has diminished in intensity.

It has recently been found by Iversen and Lenstrup* and by Howland and Kramer† that there is a characteristic chemic alteration of the *blood* in rickets. The percentage of inorganic phosphate is diminished.‡ This observation is in harmony with that of Hess and Lundagen,§ that there is a seasonal wave of inorganic phosphate of the blood in bottle-fed infants—an ebb in the winter and early spring, and a flood in the summer—a wave which runs parallel with the seasonal curve of the incidence of rickets (Figs. 166, 167). This low content of inorganic phosphate in the blood is of value in the diagnosis of rickets as it comes about relatively early; at the present time its significance is not determined. The calcium of the blood is normal, or but slightly diminished in amount.

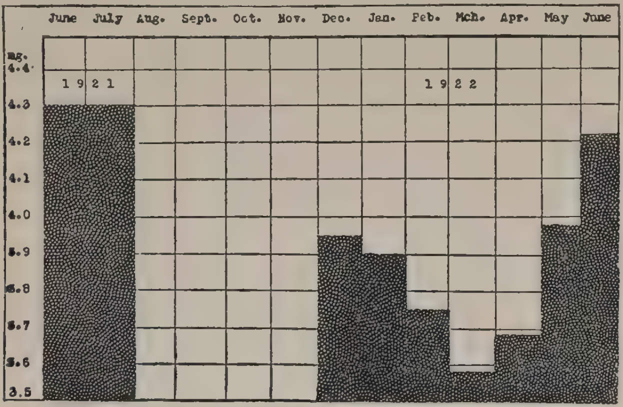

FIG. 166.—Seasonal tide of blood phosphate (inorganic).

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* Iversen, P., and Lenstrup, E., Forhandl. v Förste Nordiske, Kongres f. Paediatri (1920) (cited by Howland and Kramer).

† Howland, J., and Kramer, B., Am. J. Dis. Child., xxii (1921), p. 105.

‡ According to the method of Bell and Doisey the blood of infants contains about 4.5 mg. per cent. of inorganic phosphate. Percentages below 3.75 mg. per cent. have almost always been associated with rickets. In severe cases the percentage may fall below 2.0 mg. per cent.

§ Hess, A. F., and Lundagen, M. A., Proc. Soc. Exper. Biol. and Med., xix (1922), p. 380.

The red cells and hemoglobin may be quite normal in rickets. Anemia is not a characteristic symptom, although as the disorder advances it is generally accompanied by a secondary anemia of varying degree. There is likewise no alteration in the white cells which is constant. When rickets is accompanied by an enlargement of the lymph-nodes, there is frequently an accompanying lymphocytosis. The so-called "von Jaksch anemia" which is often associated with rickets has a typical blood picture. In this form of anemia there is a marked enlargement of the spleen.

Splenic enlargement is not, however, a sign of rickets; in mild and uncomplicated cases the spleen is not more often palpable than in infants free from rickets. Enlargement of the liver also plays no rôle in the symptomatology. It is generally stated that disturbances of the digestive tract precede the development of rickets This has not been my experience. There is more often constipation than diarrhea, and an alkaline than an acid stool. As mentioned above, diarrhea tends to mitigate the symptoms; beading of the ribs may diminish or become more angular within a fortnight following a severe attack of diarrhea.

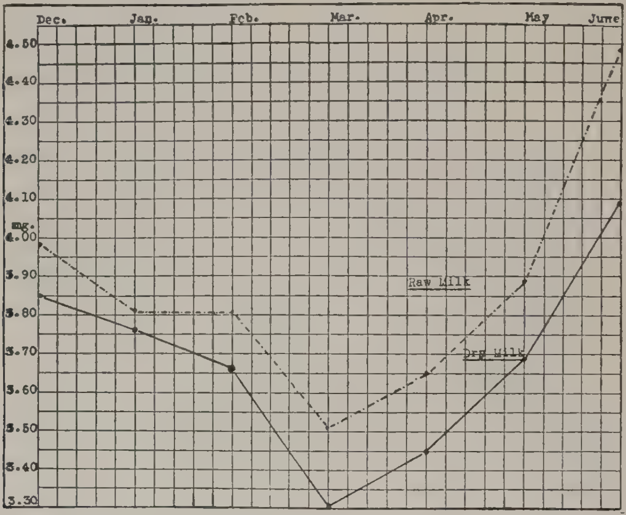

Fig. 167.—Seasonal tide of blood phosphate; diets, raw milk and dried milk.

It is an observation of long standing that rachitic children are predisposed to infections of the *respiratory tract*, in fact this is one of the chief dangers of the disorder. This seems to be true, although its explanation is obscure. Attacks of bronchitis are frequent, which might be expected as February and March are the months of greatest incidence of both respiratory infections and rickets. Pneumonia may follow and lead to death.

The *heart* is generally normal on clinic examination, although it may be found enlarged at necropsy. The deformity of the chest sometimes renders it difficult to outline its borders. The kidneys are unaffected, and no characteristic changes have been observed in the urine.

There is frequently muscular weakness. The muscles of the legs may be either undeveloped and soft, or may be large and flabby; this is true also of the muscles of the arms. The muscles of the back are unable to support the trunk when the body is placed in the sitting posture. The abdominal muscles frequently give marked evidence of the tendency to muscular weakness. There is diastasis of the recti muscles, at times accompanied by umbilical hernia, and a bulging of the lateral abdominal walls when the infant attempts to raise itself from the bed or table. If spasticity exists, the muscles of the extremities may give the impression of being firm and of good texture. Weakness may be so extreme that the infant is hardly able to move its limbs, and presents the appearance of being paralyzed.

It has long been known that rickets is a potent cause of malformation of the pelvis, which in later life leads to difficult childbirth. There are no observations to inform us as to how severe the rachitic process must have been to lead to this important condition. A recent article by Ashby* reports measurements of the pelves of 300 girls between the ages of 1 and 14. One-half of this number had suffered from rickets. The main difference between the two groups was a comparative reduction of the external conjugate measurement among the rachitic children.

As is well known *tetany* frequently is associated with rickets, and has a similar seasonal incidence. It is characterized by a hyperirritability of the nervous system which leads to convulsions, and to increased mechanical and electrical excitability of the nerves. There is also a characteristic diminution of the calcium content of the blood. It has been recently suggested by Park and his associates that tetany should be regarded merely as another form of rickets—one involving the calcium rather than the phosphorus metabolism.

In severe tetany generalized convulsions supervene. They may be infrequent or persist for so long a period as to suggest a status epilepticus; the differentiation between the two conditions will have to be made by a consideration of other symptoms and attendant circumstances. Frequently the convulsions of tetany are brought about by the onset of some infectious disease.

The other signs which should be looked for are carpopedal spasms, the Chvostek and Trousseau signs, galvanic hyperexcitability and lowered calcium in the blood. The carpopedal spasm is characteristic of tetany, and often is the first sign to suggest its presence. The hands are flexed and rotated toward the ulna side; the fingers assume the well-known "accoucheur" or "obstetric" position. The feet are extended and the toes sharply flexed. There may be other spasms. The most common are slight, evanescent twitchings of the mouth or the eyelids and laryngospasm. The latter leads to crowing inspiration in its milder form, but may reach such a degree as to endanger the life of the child by suffocation.

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* Ashby, H. J., Brit. Med. J., No. 3228 (1922), p. 905.

The Chvostek sign consists of a sudden contracture of the facial muscles on tapping the facial nerve (below the posterior part of the zygoma). Although convincing in some cases, in others it is difficult to interpret. The Trousseau phenomenon is still less satisfactory as it is more often absent than present. To elicit this sign the upper arm is constricted sufficiently to obliterate the pulse; this brings about the typical carpal spasm of the hand.

The galvanic hyperexcitability, or Erb's sign, depends on the fact that in tetany the nerves respond to a strength of current to which normally they do not react. The median, or the peroneal nerve, is tested, and the amount of galvanic current measured by means of a milliampere meter. Considerable experience is required in order to obtain reliable results. The chief abnormality consists in a cathodal opening contraction with a current of less than five milliamperes. This reaction is characteristic of tetany. Anodal hyperexcitability may likewise be present but has not the same diagnostic significance; under these conditions less than five milliamperes suffices to cause muscular contractions on anodal opening.

Normal infants have from 10 to 11 mg. of calcium per 100 c.c. of serum. In tetany this percentage is always reduced and may fall to less than half the normal. This chemical change is of great interest and clinic importance, but does not seem to be fundamental in the pathogenesis of the condition.

A mild degree of *hydrocephalus* is frequently present in rickets, in fact a large head has been regarded as one of the characteristic clinic phenomena. It is difficult to differentiate this rachitic hydrocephalus from mild degrees of non-rachitic hydrocephalus, except by means of an examination of the blood, and a consideration of other signs. Accompanying the enlargement there is the usual dilation of the superficial cranial vessels and the large fontanelle.

Relation to Other Diseases.—There has always been a great deal of discussion as to the relation of *syphilis* to rickets, some indeed, for example: Parrot, have believed that rickets is largely of syphilitic origin. The comparative incidence of syphilis and of rickets, however, precludes such a relationship, as well as the specific curative power of cod-liver oil. Clinically, there is some resemblance between the two disorders, an aspect which will be discussed below in considering the differential diagnosis of rickets. Syphilis brings about changes in the epiphyses which may readily be confused radiologically with rickets.

It is generally assumed that there is a close interrelationship between rickets and scurvy, that the one tends to lead to the development of the other. This has not been my experience, and I believe this conception is based on the occurrence of both disorders simultaneously but quite independently. The fact that orange juice is of no value whatsoever in the prevention or cure of rickets, and that, on the other hand, the use of cod-liver oil is equally futile in cases of infantile scurvy, indicates the sharp and definite distinction between these conditions. The conception of "scurvy rickets" may be compared to the equally fallacious conception of "typhoid malaria." In one respect there is an antagonism between these disorders—scurvy is associated with a cessation of growth, whereas rickets is furthered by active growth. It may be added that experiments on animals have shown that rickets cannot be brought about in the guinea pig, which is the prototype for experiments on scurvy, and that, on the other hand, scurvy cannot be induced in the rat, which is the prototype for the study of experimental rickets.

Little need be stated concerning the relationship of rickets and other diseases, excepting that, in general, conditions associated with rapid growth tend to the development of rickets, whereas those leading to a diminution of growth tend in the other direction. Well-known examples of this principle, are the babies which grow fat and heavy on a carbohydrate diet, and, on the other hand, the atrophic infant which fails to gain or thrive, but does not develop rickets. In my experience cretins do not commonly develop rickets; it is impossible to state whether this is due to lack of growth, or is directly connected with hypofunction of the thyroid gland. A relationship has been suggested between rickets and tuberculosis but has never been established clinically or experimentally. The same is true regarding rickets and malaria. In some of the older textbooks the statement will be found that rickets at times induces epilepsy. This is evidently based on the fact that tetany is closely associated with rickets and is frequently accompanied by convulsions. Such convulsions have nothing in common with those of epilepsy, nor is there any reason for believing that the irritability of the nervous system evidenced in tetany predisposes to epilepsy in later life.

Pathology.—Before the investigations of Virchow, it was believed that the essential lesion of rickets was an increased destruction or disintegration of bone. Virchow showed, however, that the chief fault lies in a lack of proper ossification. In 1885 Pommer* thoroughly elucidated the physiology of osseous tissue, rendering it evident that there is an active absorption taking place normally in growing bone, and that this process is not intensified in rickets. According to Pommer the distinguishing characteristic in rickets is that the bone which is newly laid down is not of normal composition, but is either an uncalcified or an improperly calcified tissue, and furthermore, that this tissue is laid down in superabundance. This is the accepted view today.

Although rickets should be regarded as a systemic disorder, and doubtless various tissues and organs undergo pathologic changes, the pathology of today is confined almost entirely to a consideration of alterations in bony structure.

Gross Pathology.—One of the earliest bones to be involved is the cranium. This is the seat of atrophic as well as hypertrophic change.

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* Pommer, G., Untersuchungen ueber Osteomalacie und Rachitis, Leipzig (1885).

When the calvarium is exposed to transmitted light the islands of craniotabes are clearly visible in the occipital or parietal bones. On the same skull symmetrical swellings or bosses may be present on the frontal, parietal or occipital bones. These elevations are developed from the outer table of the skull, and at the outset have a red, highly vascular interior, which later becomes organized into gray, avascular tissue. The head may be larger than normal, but the ventricles contain an excess of fluid (mild hydrocephalus). The fontanelle is open abnormally wide and its edges are thin and yielding. The rachitic craniotabes may readily be confused with the osteoporosis of the newborn or atrophic areas of syphilitic origin.

The thorax is the seat of marked rachitic changes. It is unnecessary to describe again in detail the various deformities which have been mentioned in considering the symptomatology. The beading of the ribs (rosary) is naturally more evident when the muscles are removed from the thoracic wall than during life. The striking feature—one which has been noted by all pathologists—is that the beading is generally more marked on the internal aspect of the thoracic wall, than on the external aspect; at times the beading may appear inconsiderable until the thorax is opened and an examination is made from within. This lack of correspondence is apt to occur in older infants, or where a transverse groove (Harrison's groove) has developed. Previous reference has been made to the occurrence of this depression, as well as to the depressions which run obliquely down the front of the chest on either side of the sternum. On closer examination of the "beads" a slight dislocation of the ends of the costal cartilages may be evident. Further study may reveal posterior nodosities along the ribs near the vertebral column; these result from partial fracture at these sites.

Although the spine is abnormal from a clinic standpoint, it is rarely found to be involved at necropsy. The vertebral ligaments may be excessively lax, as is true regarding any of the ligaments in the body.

The long bones are usually affected except in very mild cases. The epiphyses of the radius and of the ulna are characteristically enlarged, especially in negro infants. Similar changes may be found at the upper epiphyses of these bones, at the upper and lower ends of the humerus, the epiphyses of the femur, and the tibia at the knee joint, or, indeed, at any epiphysis. The shafts of the bones are frequently bent, especially those of the lower extremities, which have had to support the weight of the body. At the site of the attachment of ligaments bony nodosities develop. Green-stick fractures are not infrequent even in cases of moderate degree. These generally involve the humerus or the radius, and may not have been suspected during life. In the lower extremities the typical deformity is the genu varum (bow legs) or genu valgum (knock-knee). Of these various malformations, the bending of the legs, the grooving of the thorax, the bossing of the skull, and a contraction of the pelvis may persist throughout life as permanent evidence of infantile rickets.

The diagnosis of rickets frequently can be made from the gross appearance of the cut surface of a long bone sectioned longitudinally. The zone of proliferating cartilage instead of being narrow, whitish and firm, is generally greatly widened, soft and bluish-red. The distinguishing and striking feature is the irregularity of the zone of calcification, which runs zigzag across the epiphysis, and has a bluish-red tint, rather than the yellowish-white color of healthy bone. The marrow is hyperaemic, dark-red, and has prolongations or papillæ running into the soft cartilage; it contains islands of cartilage and grayish lines of osteoid. Between the cartilage and the bone is a zone of grayish osteoid, which varies greatly in width. This is the so-called metaphysis which leads to the enlargement of the epiphyses so evident during life.

Enlargement of the heart occurs in the course of rickets. This merits attention in view of the cardiac enlargement accompanying infantile beriberi and scurvy. The spleen is sometimes enlarged, as is common in infancy, but this hypertrophy is not essentially rachitic in origin.

The lungs may suffer from the pressure of the thoracic wall. Accordingly areas of collapsed lung are found subjacent to the depressions of the ribs; these are associated with areas of compensating emphysema, especially along the anterior border where the projection of the sternum removes all pressure. In severe cases more extensive collapse may be found. Bronchitis is a frequent accompaniment, and may lead to pneumonia and bring about a fatal outcome.

The *microscopic study* of rickets is of especial interest in giving information regarding the period when pathologic changes first occur. In considering this aspect, it must be borne in mind that a study of this kind relates only to lesions of the osseous system, and does not reveal rickets viewed as a systemic nutritional disorder. About 50 years ago many French authorities, as well as Kassowitz, were of the opinion that congenital rickets is of frequent occurrence. This conception was based on misinterpretation of beading of the ribs in the newborn, as well as of the areas of softening of the cranial bones. Pommer* showed definitely, however, by microscopic examination that these lesions are rarely rachitic in nature. Wieland's† intensive study of the skull bones also proved that the areas of softening are osteoporotic and not rachitic, according to our present conception of these terms. Later, Schmorl,‡ in a comprehensive examination of the bones of over 100 newborn and premature babies, failed to find rickets in any instance. These investigations do not exclude the possibility of congenital systemic rickets, but demonstrate that, as viewed from a morphological standpoint, congenital rickets must be exceedingly rare.

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* Pommer, G., "Untersuchungen ueber Osteomalacie und Rachitis," Leipiz, 1885.

† Wieland, E., Ergebn. d. inn. Med. u. Kinderh., vi (1910), p. 64.

‡ Schmorl, G., Ergebn. d. inn. Med. u. Kinderh., iv (1909), p. 403.

The first systematic pathologic study of the occurrence of rickets was made some years ago by Schmorl, who carried out routine microscopic examinations on all infants, from birth to four years of age, which were necropsied in his laboratory. This study has the advantage of having been made on unselected cases; it has the disadvantage, however, of comprising a special group of children—those of the poorest class of the city (Dresden). Furthermore, no information is furnished regarding the clinical signs in these cases, nor of the diet; it was before the day of the employment of the Roentgen-ray, or of chemical examination of the blood in the diagnosis of rickets. The most significant feature in relation to this investigation is that over 96 per cent. of children between the ages of 4 and 18 months showed signs of rickets. It should be remarked, however, that the percentage varied greatly from year to year. In no instance was rickets found during the first month of life, the earliest case occurring about the middle of the second month. Two of the tables accompanying this important investigation are reproduced (Tables 4 and 5). It will be noted that in infants two to three months of age 60 per cent. showed evidences of rickets, and 97 per cent. of those between the ages of four and six months. This is a far earlier onset than clinic observations would lead us to expect. When we consider that lesions of the bones cannot represent early rickets in its strictest sense, it must be realized that the onset is considerably earlier than is generally stated, that its beginnings are certainly in the first half rather than in the second half of the first year of life. The second table (Table 5) indicates the span of time included in "beginning rickets of the bones." It may be noted that Schmorl found cases to begin not only as early as the second month, but some to have their onset as late as the end of the second year. This period corresponds with clinic experience. The table shows us, furthermore, that florid rickets, in other words, rickets without tendency to healing, occurs even toward the end of the fourth year.

TABLE 4.—DISTRIBUTION OF RICKETS CASES ACCORDING TO AGE (SCHMORL)

Number of children	Age, months	Total number of cases		Rickets absent	
		Cases	Per cent.	Cases	Per cent.
33	2–3	20	60.6	13	39.4
34	4–6	33	97.0	1	3.0
53	7–9	50	94.0	3	6.0
75	10–12	73	97.3	2	2.7
59	13–18	58	98.3	1	1.7
33	19–24	30	90.9	3	9.1
65	25–36	57	87.7	8	12.3
34	37–48	24	70.6	10	29.4

Table 5.—Distribution of Stages of Rickets According to Age (Schmorl)

Ages, months	Beginning		Florid		Healing		Healed		Total
	Cases	Per cent.	Cases	Per cent.	Cases	Per cent.	Cases	Per cent.	
2	4	100.0	..	....	..	....	..	....	4
3	12	75.0	4	25.0	..	....	..	....	16
4–6	19	57.6	7	21.2	7	21.2	..	....	33
7–9	17	34.0	23	46.0	10	20.0	..	....	50
10–12	7	9.5	45	61.7	20	27.4	1	1.4	73
13–18	4	6.9	32	55.2	15	25.8	7	2.1	58
19–24	2	6.6	10	33.4	11	36.6	7	23.4	30
25–36	..	.....	13	22.9	14	24.5	30	52.6	57
37–48	..	.....	2	8.3	3	12.5	19	79.2	24

The incidence of rickets according to season, and month by month, is given in detail and is represented by the seasonal curves which are reproduced in Fig. 159. On studying critically Schmorl's figures on which these curves are based, certain fallacies become apparent as to the use of pathologic data in judging of phenomena associated with rickets. According to these figures "beginning rickets" tends to become less frequent after the month of January; the incidence of "florid rickets" does not decrease until June; "healing rickets" does not increase markedly until the month of July. In each instance the alteration is unduly delayed, which indicates that microscopic change in the bones is not a sufficiently prompt and sensitive reagent to serve as a criterion of the beginning of the development, or of the healing of this disorder.

The chief effect of the rachitic process is to produce bone which is abnormally soft. This is evident macroscopically on cutting the bone, and microscopically by its lack of calcium and irregular structure. The significant feature is the development of excess of osteoid tissue, tissue which is imperfectly calcified (Figs. 168, 169). The osteoid may be formed from the marrow within or from the periosteum without. Normal bone has a narrow border of osteoid surrounding the trabeculæ of the spongiosa. In rickets this is exaggerated, so that in extreme instances it may almost fill the marrow cavity. In such cases the marrow cells are few in number, and are replaced by spindle-cells and exuberant blood-vessels, osteoblasts being present in varying numbers. The osteoid developing from without, springing from the periosteum, may give rise to irregular masses between it and the underlying bone. It may be present in this form—the so-called periosteal callus—or in definite layers which can be separated individually, or as small hard osteophytes. In separating the thickened periosteum, which can

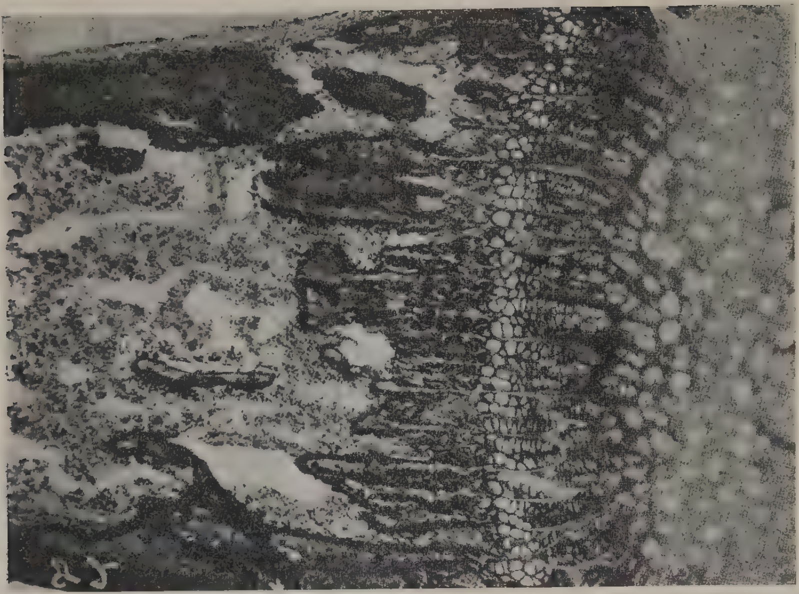

FIG. 168.—Normal rib. Rat weight 158 gm. 52 days on complete diet; continuous growth and gain in weight. Decalcified three days in Müller's fluid.

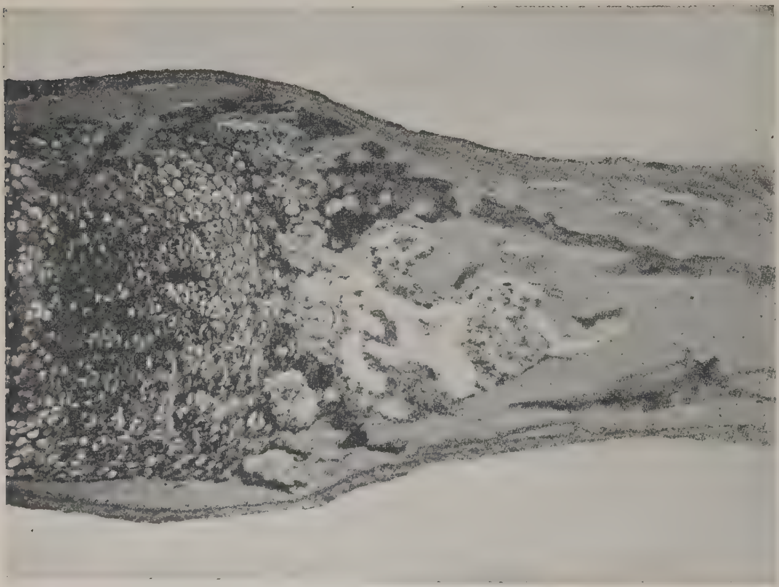

FIG. 169.—Rib of rat. Thirty-four days on rickets-producing diet. Marked rachitic lesions. Note increased width and irregularity of proliferative cartilage, absence of calcium deposition, great excess of osteoid in region of metaphysis and about cortex. Decalcified five days in Müller's fluid.

readily be torn from the cortex, pieces of the soft bone frequently adhere to its inner surface.

The disturbance in endochondral ossification is especially marked, leading to a confusion of the various layers, which intermingle with each other. One observes a marked increase in the cartilaginous zone, which may be many times its normal width. As one proceeds toward the diaphysis from the resting cartilage, one notes how the cells increase in size, gradually forming large columns containing an immense number of cells. Proceeding further we find the columns disappearing and replaced by large isolated cartilage capsules. In this zone of uncalcified cartilage, scattered here and there, large islands of marrow are to be seen, highly vascular and in some instances associated with osteoid.

Adjacent to the cartilaginous layer is the typical osteoid layer—the rachitic metaphysis—a tissue of varying width. Here the abnormal bone (osteoid) is found in irregular formation. Among this tissue there are wide spaces containing marrow and many spindle-cells. In this area there are also islands of uncalcified cartilage, which in severe cases may be sufficiently large and prominent to be distinguished by the naked eye. The next zone shows osteoid which has been formed mainly from the marrow but partly directly from the cartilage, and which is impregnated with calcium. The marrow itself is highly vascular, a condition which Kassowitz interpreted as inflammatory, and which he regarded as the primary lesion of rickets.

This typical arrangement of rachitic zones is frequently altered by the substitution of connective tissue and uncalcified bone in place of the superabundant cartilage. Schmorl has attributed this formation of connective tissue to mechanical influences, and ascribed the fibroid tissue so frequently found at the junction of the epiphyses and the diaphyses of the ribs, to exceptional pressure at this point. If the trauma is sufficient a tear may result with extravasation of blood and a partial dislocation at the costochondral junction.

The healing of rickets is an interesting process, especially as it relates to the cartilage. The first sign is the development of a zone of preparatory calcification. This does not take place, as in normal endochondral ossification at the junction of bone and cartilage, but within the cartilaginous zone itself. A thin, more or less irregular, transverse stripe is formed below the resting cartilage, which may be apparent macroscopically as a yellowish-white line running across the hypertrophied cartilage. This calcification occurs at the site where it would normally have taken place had the rachitic process not intervened. As the healing process advances the cartilage is absorbed and replaced by many small trabeculæ of bone, so that we may see side by side, in irregular formation, the cartilage of the florid rachitic period and the newly laid down bands of bone.

The healing process is frequently interrupted by relapses; according to Schmorl's experience this occurs in 40 per cent. of cases. Under these circumstances, the newly formed zone of preparatory calcification becomes partially absorbed, so that its course is broken, or only vestiges are apparent along the sides of the bone. On the other hand, it frequently does not undergo absorption but is pushed away from the epiphysis by the new formed tissue. The relapse may be in turn succeeded by a remission; indeed these alternations of healing and of fresh rachitic invasion may follow each other several times. In such cases if the zone of calcification remains intact we find one layer overlying the other. Under such circumstances the most recent layer is the one situated nearest the epiphysis and *vice versa*. It is not known how rapidly this calcification of bone can take place. After giving cod-liver oil, or ultraviolet therapy to rachitic rats, Pappenheimer* recently has shown that (following a single dose of cod-liver oil), healing could be noted within 24 hours. He summarizes the evidences of healing as follows: (1) Deposition of calcium in the distal half of the widened zone of preparatory calcification; (2) invasion of the calcified portion from the lateral and subchondral sides by large blood-vessels, with destruction of the cartilage cells; (3) changes in the osteoid tissue which may be taken to indicate beginning lysis or halisteresis; (4) great dilatation and congestion of the blood sinuses in

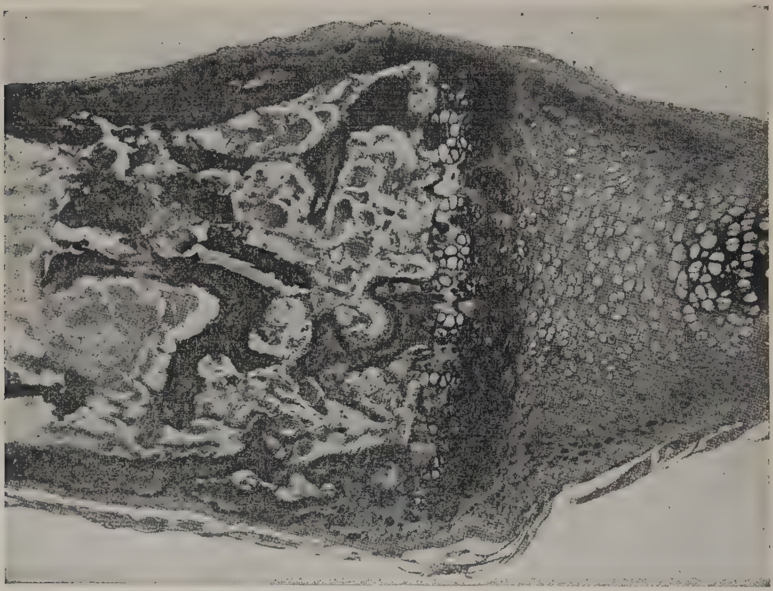

Fig. 170.—Rib of rat. Eighty-two days on fat-soluble A deficient diet. Maximum weight 140 gm. Terminal weight 125 gm. Decalcified five days in Müller's fluid.

Zone of preparatory calcification not over four cells in depth, regular, matrix completely calcified. Osteogenesis at epiphysis is fairly active. Spongiosa composed of coarse trabeculæ, with absent or inconscpicuous osteoid margin. Cortex broad and completely calcified. Marrow cellular, not fibrous.

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* Pappenheimer, A. M., J. Exper. Med., xxxvi (1922), p. 335.

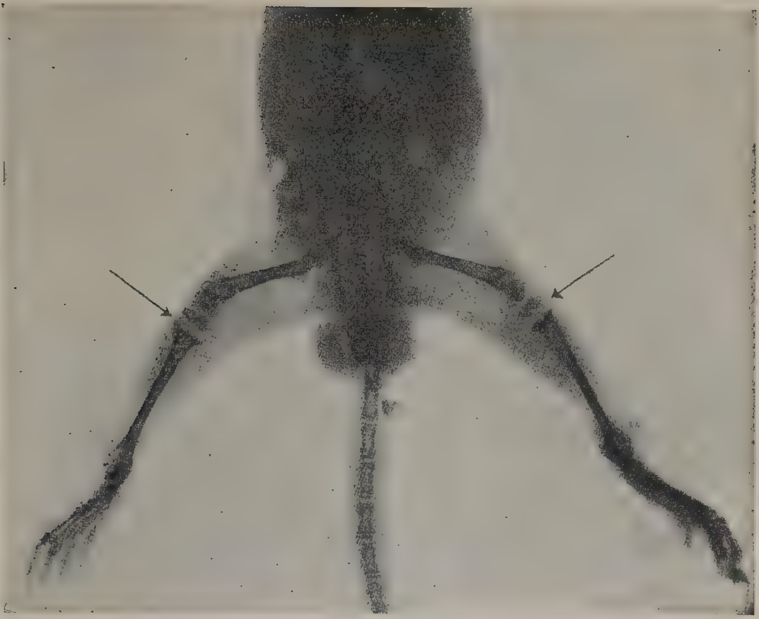

FIG. 171.—Rat forty-one days on rickets-producing diet. *X*-ray, showing rachitic changes in upper epiphysis of tibia.

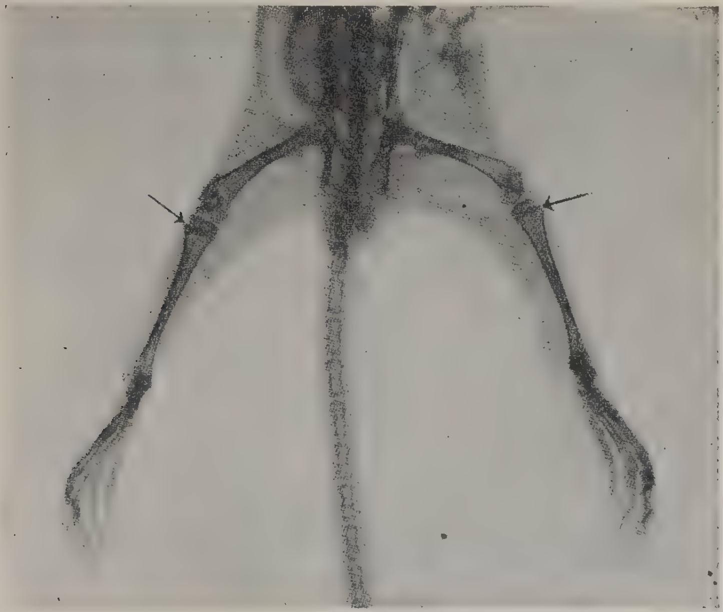

FIG. 172.—Rat 175 days on fat-soluble A deficient diet. *X*-ray. Absence of rachitic lesions at upper epiphysis of tibia.

the metaphyseal region; and (5) deposition of granular calcium in the perichondral and pericortical osteoid.

The relationship of osteoporosis and rickets has been the subject of long discussion and is still undecided; some believe these lesions to be pathogenetically distinct, whereas others are of the opinion that they represent merely variations of the same essential process. In infants osteoporosis is frequently found in the skull bones at, or soon after birth. It is also present in the long bones of atrophic infants. In puppies it has been brought about by a lack of calcium in the diet. When rats are deprived of the fat soluble vitamin they develop osteoporosis and not the typical rickets associated with an increase of osteoid.* Thus pathologic investigation confirms the clinic experience that this nutritional factor is not the "antirachitic vitamin" and that rickets cannot be ascribed to its deficiency in the diet.

The histologic picture of osteoporosis brought about by a dietary deficient in this factor was as follows: The zone of proliferating cartilage was narrow, and its matrix invariably densely calcified. The primary spongiosa was represented by a few delicate and irregular disposed trabeculæ. The osteoblasts surrounding these trabeculæ were inconspicuous in contrast to their swollen appearance in actively proliferating bone. Osteoid tissue was conspicuously absent. It is clear that these changes in no respect resemble those of rickets (Figs. 170–172).

Diagnosis and Differential Diagnosis.—For a variety of causes rickets is more often overlooked than diagnosed. First among these sins of omission is the failure to examine for rickets, the tendency to ignore its existence unless the signs are so evident as to force themselves on the attention. It is a disorder which is readily passed over, even when developed to a moderate degree. Furthermore, the technique of examination is frequently faulty. In examining for beading of the ribs, the thorax is palpated either too far anteriorly or posteriorly, the craniotabes is not discovered, and slight degrees of various other signs pass unnoticed. Added to these difficulties there is the inherent obscurity of symptoms associated with the diagnosis of all nutritional disorders, which renders it almost impossible to recognize them in their early stages.

Of the direct clinical signs the greatest stress, in my opinion, should be laid on the beading of the ribs (Fig. 173). After one has learned from experience how great a degree of beading may be found unassociated with rickets, this sign acquires definite value. A rosary of moderate size, of secondary degree, is almost diagnostic of rickets. Craniotabes is also valuable in establishing the diagnosis, but unless the course of the softening has been followed for several months, it is oftentimes difficult to differentiate it from the craniotabes encountered so frequently in the newborn and in very young infants. In fact, the one may merge into the other, and as the latter tends to disappear, the former may develop.

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* Hess, A. F., McCann, G. F., and Pappenheimer, A. M., J. Biol. Chem., xlvii (1921), p. 395.

The radiograph is of great aid in diagnosis. It should be borne in mind, however, that the rachitic process must have been developing for some months before it becomes apparent by the Roentgen-ray. Nevertheless, bony lesions cannot have advanced far before they are manifest by this means. To a certain extent the roentgenologic picture is a subjective rather than an objective sign, for in its minor manifestations it is open to individual interpretation. For example: It is difficult to appraise the importance or significance of slight spreading of the lower epiphyses of the ulna, an alteration which is frequently seen and which may or may not advance further. Spreading of the lower end of the ulna, accompanied by concavity or cupping of its border, associated as it generally is with irregularity or haziness of outline is characteristic. The shaft of the bone often shows a deficiency of inorganic salts which allows the organic frame work to stand out with abnormal detail. Similar changes are to be found at the knee joint in the epiphyses of the femur, and less often in the extremities of other long bones (Figs. 163, 165).

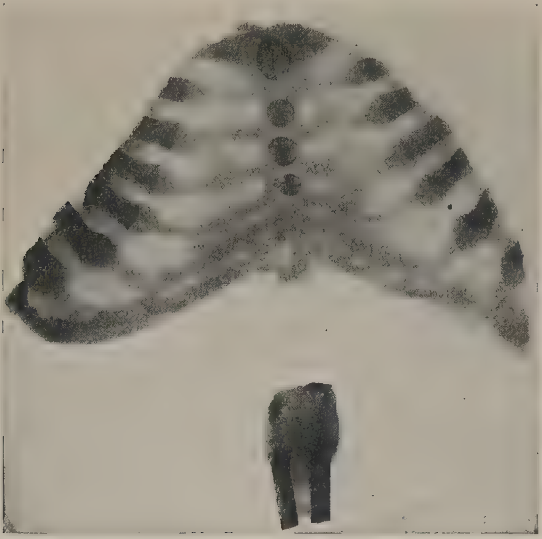

Fig. 173.—Postmortem radiograph of costochondral junction and of epiphyses of wrist; showing marked beading of the ribs coincident with normal epiphyses of the radius and ulna.

At the present time it is difficult to evaluate the diagnostic importance of a diminution of inorganic phosphate in the blood. From a practical standpoint the diagnosis will, in the great majority of cases, have to depend upon simpler and more available signs. It is possible, however, that a chemical examination of the blood will enable us to determine the period of metabolic changes in rickets before the solid tissues are altered, lesions which are rather the result of this disorder than evidences of the underlying disturbance. The normal inorganic phosphate of the blood of infants is about 4 to 4.5 mg. per 100 c.c. of blood or plasma. In active rickets the percentage falls to 3 or 3.5 per cent., or even lower in the severe forms.

In the early diagnosis delayed eruption of the teeth, pot-belly, diastasis of the recti muscles, delayed standing or walking, have been stressed far too much. Sweating of the head is a valuable early sign, but one which is suggestive rather than diagnostic, and should stimulate the search for further evidences of this disorder.

The signs of advanced rickets are too well known to require more than passing mention. The bowed legs, the knock knees, the square bossy head, the deformed thorax, have been discussed in considering symptomatology. Whether bowed legs are always due to rickets is by no means certain; it is a deformity which may appear after all other signs have disappeared, or at times in overweight infants who have manifested no previous signs of rickets.

It may be well to emphasize again the fact that rickets is not associated with malnutrition, that it should be looked for in the well-nourished, apparently normal infant, and that breast feeding does not by any means exclude its development or presence.

In the differential diagnosis of rickets the main diseases to be borne in mind are infantile scurvy and congenital syphilis. The former is no doubt frequently confused with rickets. This confusion extends to the therapy of the two disorders, and one frequently finds that orange juice is considered of value in the treatment of rickets. In the diagnosis between these two conditions, the history of the previous dietary, the clinic signs and the therapeutic test are all of importance. For the development of scurvy the infant, in almost all instances, must have been deprived of fruit juices and of vegetables, and have been artificially fed on milk which has been pasteurized or boiled. The scorbutic baby is generally poorly nourished, unhappy, capricious in its appetite, and tender to the touch. When its thorax or the lower ends of its thighs are pressed, it shows distinct evidence of pain. Petechiæ may be present on the skin, or red blood-cells in the urine. The inorganic phosphate of the blood is normal, and the radiographic picture is either negative or shows the "white line" of Fraenkel at the epiphyses. Beading of the ribs may be present, and is more angular and less rounded in character. It is this "scorbutic beading" which has led to a confusion of the two disorders and to the opinion that they are generally associated. In my opinion rickets does not tend to the development of scurvy, or scurvy to rickets. From a pathologic standpoint they have nothing in common.

Fournier believed that rickets was due to syphilis, and Marfan today accords syphilis an important etiologic rôle. Undoubtedly similarities exist. The large square head is found in both, but is more marked in syphilis, and is associated with larger frontal bosses. Both may manifest curvature of the long bones, dwarfing in stature, craniotabes. It is on the positive signs of syphilis which stress must be laid in differentiating the two conditions. In syphilis the Wasserman reaction may be of decisive value, although it may fail us in young infants. However, the characteristic facies, the rash, the bilateral enlargement of the epitrochlear glands, the enlarged, hard spleen, the periosteal swelling, will almost always enable one to differentiate. The radiographic picture may prove somewhat confusing. Where the lesions, as noted by the Roentgen-ray, are not improved by two or more months of treatment with cod-liver oil, the existence of syphilis should be suspected. In cases of doubt an estimation of the inorganic phosphate of the blood is of value, as this constituent is not reduced in congenital syphilis.

Mention has been made of the occurrence of the square head of congenital syphilis, and of "the rosary" in scurvy. There are some other bony lesions of rickets which at times present difficulty in diagnosis. Pott's disease or tuberculosis of the vertebræ occasionally offers difficulties; the main distinctions are that in rickets the kyphosis is less marked, that it is not tender, that it is readily rectified by placing the infant on its abdomen, and that it is more diffuse and less localized to one or two vertebræ. Although these criteria serve generally to differentiate between the two conditions, it may be necessary to search for other signs of syphilis or of rickets in order to come to a satisfactory determination.

In severe cases coxa vara may result from rickets and be confused with congenital dislocation of the hip joint. Under such conditions a careful examination of the acetabulum combined with radiographs of this area, and comparative measurements of the lower extremities will serve to establish a differentiation.

Rachitic fractures of the long bones may be diagnosed as the syphilitic pseudo paralysis described by Parrot. The main distinction between these conditions is a chronologic one, the syphilitic lesion occurring during the first two or three months, in other words at a period far too early for rachitic fractures. The Roentgen-ray as well as a consideration of systemic signs and symptoms, should prevent a confusion of this kind.

Prognosis.—The prognosis of rickets is generally very favorable. The majority of cases are very mild, so mild indeed that many pass unrecognized and leave no local deformity or systemic disturbance. Recovery comes about spontaneously as the result of several factors. First among these must be reckoned the fortuitous advent of spring or summer, with its sunshine and other favorable climatic influences; furthermore, the decrease in the growth rate which occurs after the first year of life tends to retard its development; the natural enlargement of the dietary, including eggs, etc., which is allowed the infant during its second year of life, induces a cessation of the rachitic process. It is indeed remarkable to observe the degree to which these "spontaneous" cures may extend. Bowed legs, beading of the ribs, enlarged epiphyses, gradually disappear and the bones regain their normal contour. The outcome may, however, not be so favorable, especially in severe cases in which almost any of the bony lesions may be permanent. The square shaped rachitic skull can frequently be recognized in the adult, or the beading of the ribs, or Harrison's groove. Less often the so-called "chicken breast," with its prominent sternum, or the "funnel-shaped chest" with its depressed sternum, is found as an aftermath of the rachitic process. These deformities are, however, not pathognomonic of rickets. Scoliosis or kyphosis of the spinal column is often the result of this disorder. The deformities associated in the lay mind most often with rickets are the bow legs and the knock knees. When developed only to a moderate degree, it is surprising how often these bends will straighten without medical intervention; the extreme forms persist throughout adult life. Other deformities of the lower extremities which may lead to permanent disability, are the coxa vara, the depression of the neck of the femur, and flat foot. The most important bony deformity due to rickets is that of the pelvic bones which, in women, leads to varying degrees of dystocia. The obstruction may be so great as to render Caesarean section necessary. For a further consideration of this aspect the reader is referred to treatises on obstetrics.

As has been mentioned, rickets renders the organism more susceptible to catarrhal infections, especially to those of the respiratory tract. Thus it may prove the predisposing cause of bronchitis or of bronchopneumonia. In the most extreme cases associated with marked deformity of the thorax—cases which are rarely seen in this country, except among the negroes—pressure may be exerted on the lungs sufficient to bring about pneumonia, atelectasis and emphysema.

It has been claimed that the heart may suffer from a severe rachitic condition. This is true to marked degree for beriberi, and to a less degree for infantile scurvy. Enlargement of the heart may result from rickets, but it is impossible to state whether this may lead to permanent weakness of its muscle or defect of innervation.*

Rickets may cause a permanent stunting of growth, but in ordre that this impairment result, the disorder must have existed for a long period and to an extreme degree. Most of the cases of rachitic dwarfism are probably due to additional factors—to complicating scurvy, to an insufficiency of the diet from a quantitative standpoint, or to intercurrent infections. According to some observations which we have made, mild rickets does not lead to growth retardation, whereas ordinary infections—if repeated—do bring about this result.

Treatment.—A consideration of the treatment of rickets is most satisfactory, as we are placed in the unique position of possessing two specific therapeutic measures—cod-liver oil and ultraviolet irradiation. However, before discussing the cure we should consider the means by which this widespread nutritional disorder may be prevented.

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* Mellanby has drawn attention to an experience which we have also noted, namely, that animals suffering from rickets have a tendency to die suddenly following ether narcosis.

Prophylaxis.—As stated in considering etiology, all agree that rickets occurs less frequently and in less severe degree among breast fed, than among artificially fed infants. Maternal nursing or woman's milk constitutes, therefore, a valuable prophylactic measure. This is, however, by no means an absolute safeguard, as mild forms of rickets are very common among thriving breast fed babies. Why this should be the case is not understood. It may be linked with a deficiency in the diet of the mother during pregnancy and lactation, a point of view which gives hope that in the future prophylaxis may be facilitated by regulating the dietary of the mother. What effect in this regard the ration of the cow has is undetermined. A food which, in my experience, has proved of value in protecting the artificially fed infant is egg yolk. Babies receiving one egg yolk daily generally have been able to go through the entire winter without showing signs of rickets, or diminution of the inorganic phosphate of the blood. The egg yolk is mixed with the milk and given in the bottle; it enriches the percentage of fat in a quart of milk by about 0.5 per cent. Egg yolk is well tolerated by almost all babies and may be given at one to two months of age. Less than an entire yolk may be given to very young infants. This food has the additional advantage of adding iron and fat soluble vitamin to the diet. The prophylactic and curative value of egg yolk has been amply demonstrated in the laboratory and the clinic.*

Hygiene plays an important rôle in prophylaxis. It is impossible to evaluate the relative importance of cleanliness, fresh air, climate and various meteorologic conditions. However, sunlight is the most important of these factors. Infants should be out of doors as much as possible and should not be shielded assiduously, as is the custom, from the rays of the sun. It is not necessary that the infant be subjected to the direct rays of the sun. Were such the case few babies would receive the benefit of sunlight. The scattered diffuse radiations contain a large amount of ultra violet and are, therefore, of protective and curative value. Moreover, the ultraviolet waves of direct sunlight are able to penetrate clothing material to a certain extent, provided it is not too thick or closely woven. Animal experiments have shown that white goods are much more permeable than black material of the same weave. During the winter the sunlight in this climate is so poor in ultraviolet rays that it is doubtful whether it alone can be relied upon for protection. Moderate altitude—3000 to 5000 ft.—seems to be more favorable in the prevention of rickets than sea level. This is true likewise of a dry climate as compared to one which is humid.

In view of the great prevalence of rickets, the question arises whether all infants should be given routine prophylactic treatment, just as fruit juice is regularly added to the diet of artificially fed babies to prevent scurvy. At present such a procedure seems advisable; it is quite possible that at some future time we shall be able to so alter the milk of the mother, or of the cow, that it will possess the necessary protective properties. The most reliable and available antirachitic is cod-liver oil. This therapeutic agent has been used in England and on the shores of the Baltic for centuries, and was introduced into France by Bretonneau about 100 years ago, and strongly recommended by his illustrious pupil Trousseau. Kassowitz of Vienna later prescribed it combined with small amounts of phosphorus, and, indeed, attributed its therapeutic value to the phosphorus rather than to the peculiar virtue of the fish oil. By some it was considered to have special value, but until recently authoritative children's specialists regarded it merely as the equivalent of other vegetable and animal oils. A careful clinic study among negroes carried out a few years ago by Hess and Unger showed the beneficent effect of cod-liver oil—rickets developing to a far greater degree among the infants who did not receive the oil than among those that did. In his first report, Mellanby was able to show by means of radiographs that calcification of the epiphyses could be brought about in puppies by small additions of cod-liver oil to the diet. It will be well, therefore, until some other form of antirachitic is established, to give small doses of cod-liver oil to infants during the winter, from October to May. Five drops of pure oil may be given three times a day at the age of one month, or 15 drops be mixed with the day's total feeding, provided the infant takes all its food. This dosage should be gradually increased so that the baby receives one-half a teaspoonful of oil three times a day when it is three months of age. A less amount probably will afford protection in most instances.* In the event that the oil is not well tolerated it may be given in orange juice, or combined with malt, or in the form of an emulsion; under such circumstances the dosage will have to be increased in order to furnish an adequate amount of the active principle. Phosphorus may be combined as follows:

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*Hess, A. F., Jour. Am. Med. Assoc., lxxxi, 1923, p. 15.

Phosphori	0.01
Ol. Morrhuæ	200.0

Although there may be a difference of opinion as to whether all infants—especially the breast fed—should receive prophylactic treatment, there can be no such difference in regard to premature infants or twins. Unless these classes of infants are specifically protected they will almost invariably develop rickets. The same holds true, to a less degree, of negro and Italian babies living in the northern part of the United States. For twins and premature infants the need of protection is so great that if the oil is not tolerated, they should receive treatment by the other specific therapeutic agent—ultraviolet rays.

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* The active principle of cod-liver oil is not known. Zucker† has recently shown that it is contained in the ether soluble unsaponifiable fraction of the oil. It is similar in its solubilities to cholesterol and highly resistant to heat and to alkalinization. He has been able to concentrate it so that it possesses many hundred times its original potency. This investigation, quite apart from its theoretical significance, leads us to hope that in the near future it will be possible to give small doses of a concentrated extract of cod-liver oil for curative or prophylactic purposes.

† Zucker, T. F., Pappenheimer. A. M., and Barnett, M., Proc. Soc. Exper. Biol. and Med., xix (1922), p. 167.

The relation of light to rickets has been mentioned in various connections. Recently it has been shown by Huldschinsky* that the radiations of the mercury vapor quartz lamp are able to protect against, or to cure rickets. Hess and Unger† demonstrated that sunlight was able to accomplish a similar result provided its effective rays were not filtered out by means of window glass.‡. They also showed that irradiation with the carbon arc lamp§ may be used for this purpose. During the winter months the sunlight is so poor in ultraviolet rays and so unreliable from day to day, that it is necessary to have recourse to artificial sources. For protection or for cure irradiation is carried out as follows. Where the air-cooled mercury vapor lamp is used it is well to have the lamp at a distance of 3 ft. from the baby, and to begin with small exposures in order to avoid superficial burns. There is a great difference in the tolerance of individuals to ultraviolet radiations. The eyes must be protected with glasses and all clothing removed. The first exposure should be for three minutes, increased by two minutes each session until the irradiation is carried out for 20 minutes. The entire body is irradiated, a different part being selected for successive treatments. Under this method of therapy the craniotabes and osteoporosis of the cranial bones—signs so commonly met with in the premature infants—rapidly disappear. Where rickets is evident in the epiphyses, radiation rapidly brings about calcification, which can be followed in radiographs. Tetany likewise yields to this treatment, and should be combined with the routine calcium therapy.

The carbon arc lamp (with white-flame carbons) may be used as the source of radiation (Table 6). This gives a spectrum more closely approaching that of sunlight. Its advantage is that it is not irritating to the skin, does not cause burns, and that longer exposures can be employed from the outset. The initial treatment should be for one-half hour at a distance of 3 ft. (Figs. 174, 175). This degree of radiation will suffice for prevention or cure, but may be safely increased to one hour. Pigmentation of the skin does not readily come about as the result of the rays from the carbon arc as is the case with the mercury vapor lamp. In institutions groups of infants may be successfully irradiated by means of a carbon arc lamp at a distance of 9 ft.; by this mm

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* Huldschinsky, K., Zeitschr. f. orthop. Chir., xxxix (1920), p. 426.

† Hess, A. F., and Unger, L. J., J. A. M. A., lxxvii (1921), p. 39.

‡ All ultraviolet rays are not of value in rickets, those longer than about $313\mu\mu$ seeming to be inert in protective or curative value. As the sun's radiations extend down merely to $290\mu\mu$, it is evident that only a small amount of its radiation is active in this respect. These facts emphasize both the specificity of the wave lengths—a difference of about 0.0000025 mm. determining whether or not radiations are potent—and also the small degree of effective rays necessary in order to prevent the occurrence of rickets. It is possible that it will be found practicable to provide for infants outdoor pavilions with windows of special glass which allow the passage of effective ultraviolet radiations. At present the cost of such glass is prohibitive for child-caring institutions.

§ Hess, A. F., and Unger, L. J., J. A. M. A., lxxviii (1922), p. 1596.

TABLE 6.—RACHITIC INFANTS TREATED WITH CARBON ARC LIGHT

Case	Age, months	Diet	Treatment			Roentgen examinations		Inorganic phosphate (blood)		Comment
			Dates	Length, minutes	Distance, feet	Dates	Rickets	Date	Mg. %	
1. M. E.	29	Malt soup	12/ 9/21	15	4	12/15/21	Moderate	12/ 6/21	3.70	K_2HPO_4, 2 gm. daily, 10/16/21 to 12/8/21
			12/16/21	30	4	1/ 2/22	Healing	1/ 5/22	4.00	
			1/ 8/22	30	3	1/17/22	Improved	2/11/22	4.36	
			to 2/22/22	30	3	2/18/22	Healed	3/14/22	4.03	
						3/18/22	Healed	4/14/22	4.00	
2. Y. D.	12	Dry milk	12/ 9/21	15	4	12/15/21	Slight	12/ 5/21	3.70	
			12/16/21	30	4	1/2/22	Healing	1/5/22	3.90	
			1/ 8/22	30	3	1/17/22	Healing	2/15/22	4.31	
			to 2/22/22	30	3	2/18/22	Healed	3/15/22	4.08	
						3/18/22	Healed	4/13/22	4.03	
3. S. L.	5½	Raw milk	1/30/22	15	3	1/17/22	Slight	1/29/22	3.84	
			2/14/22	30	3	2/18/22	Healing	2/28/22	4.34	
			to 3/21/22	30	3	3/18/22	Healed	3/27/22	4.16	
						4/15/22	Healed	4/27/22	4.16	
4. D. R.	13	Raw milk	1/30/22	15	3	1/17/22	Moderate	1/26/22	3.49	
			2/14/22	30	3	2/18/22	Healing	2/28/22	4.16	
			3/21/22	30	3	3/18/22	Healed	3/27/22	4.16	
						4/15/22	Healed	4/27/22	4.27	
5. C. B.	6½	Raw milk	2/ 6/22	120	9	1/17/22	Slight	1/26/22	3.69	
			to 3/ 5/22	120	9	2/18/22	Healing	2/28/22	4.29	
						3/ 4/22	Healed	3/27/22	4.25	
						3/ 8/22	Healed	4/27/22	4.36	
						4/15/22				
6. M. C.	9	Dry milk	2/ 6/22	120	9	1/17/22	Slight	2/ 4/22	3.84	
			3/ 5/22	120	9	3/18/22	Healing	2/ 3/22	4.44	
						3/ 4/22	Healed	4/ 3/22	4.00	

						3/18/22	Healed	5/ 3/22	4.54	
						4/15/22	Healed			
7. R. S.	14	Dry milk	2/19/22	120	9	2/18/22	Moderate	2/17/22	3.73	
			to 5/ 9/22	120	9	3/ 4/22	Moderate	3/17/22	4.34	
						3/17/22	Healing	4/14/22	4.00	
						4/ 1/22	Improved	5/10/22	4.54	
						4/15/22	Almost healed			
8. F. F.	12	Protein milk	2/19/22	60	9	2/18/22	Moderate	2/ 9/22	3.71	
			to 5/ 1/22	60	9	3/ 4/22	Moderate	3/10/22	3.77	
						3/17/22	Healing	4/10/22	4.16	
						4/ 1/22	Improved	5/10/22	4.20	
						4/15/22	Improved			
9. D. J.	14	Dry milk	3/ 7/22	60	9	3/ 4/22	Moderate	1/ 7/22	3.11	K_2HPO_4, 2 gm. daily,
			to 5/ 9/22	60	9	3/18/22	Healing	2/ 3/22	3.22	1/3/22 to 3/5/22
						4/ 1/22	Improved	3/ 3/22	2.77	
						4/15/22	Almost healed	4/ 1/22	3.57	
								5/ 2/22	4.46	
10. H. B.	17½	Raw milk	3/ 7/22	120	9	3/ 4/22	Marked	12/30/21	2.94	K_2HPO_4, 2 gm. daily,
			to 5/ 9/22	120	9			2/ 5/22	3.11	1/3/22 to 3/5/22
						3/17/22	Healing	3/ 5/22	2.17	
						4/ 1/22	Improved	4/ 2/22	3.44	
						4/15/22	Improved	5/ 9/22	3.75	
11. P. B.	14	Protein milk	3/23/22	60	9	2/18/22	Slight	....	3.59	K_2HPO_4, 2 gm. daily,
			to 5/ 9/22	60	9	3/18/22	Moderate	3/17/22	3.57	2/23/22 to 3/23/22
						4/ 1/22	Improved	4/14/22	3.92	
						4/15/22	Improved	5/10/22	4.34	

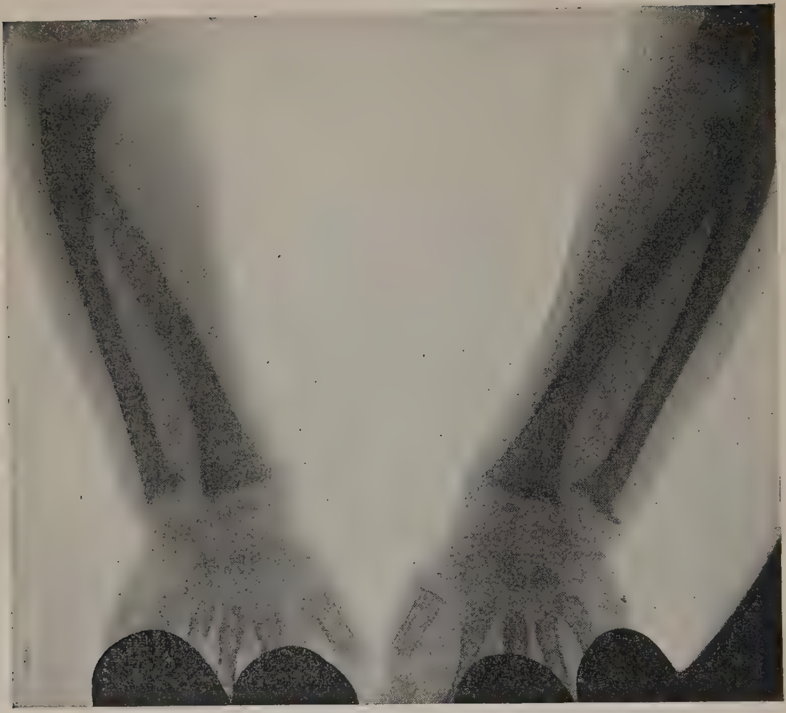

Fig. 174.—Moderate rickets. Dec. 15, 1921.

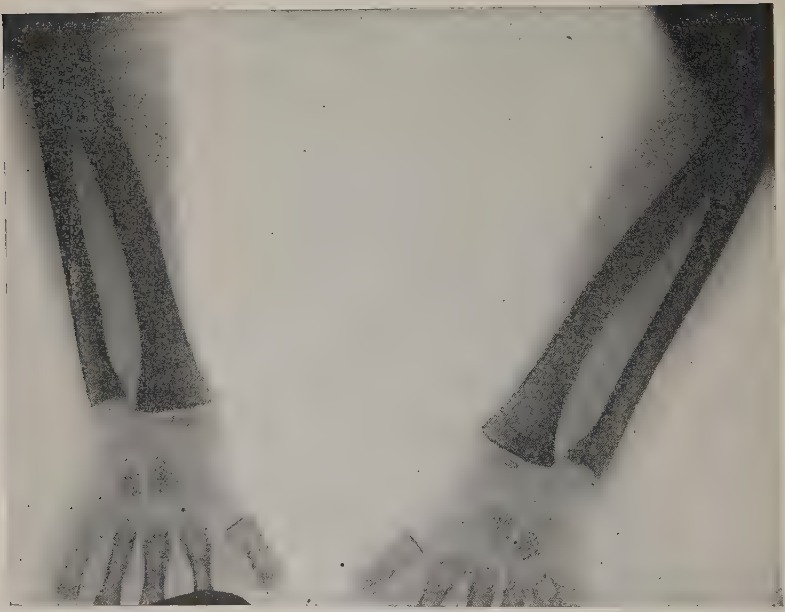

Fig. 175.—Feb. 18, 1921. Same case as Fig. 174. Healed rickets. Daily irradiation for one-half hour with carbon arc lamp.

method we are provided at one and the same time with both illumination and antirachitic therapy.

In the cure of rickets we have at our disposal the two specifics which have been discussed in relation to prophylaxis. An infant six months old or more, suffering from rickets, should receive a teaspoonful of the pure cod-liver oil three times a day. It is well to begin with half this amount, increasing gradually until full dosage is attained. Some infants do not tolerate cod-liver oil in any form; some will tolerate the more palatable emulsions. Except in extreme cases it is not necessary to give the oil during the summer months as sunshine and other climatic factors suffice to bring about a rapid cure.

Where cod-liver oil is not tolerated, or is undesirable, radiotherapy may be employed. When both measures are combined it is remarkable to observe the rapid calcification of the epiphyses; 10 days may suffice to give evidence of improvement. For this purpose, we may resort to the mercury vapor quartz lamp (air-cooled), the carbon arc lamp, or to direct sunshine. The technique of using the lamps is identical with that in relation to prophylaxis. The difficulty with heliotherapy is inherent in the uncertainty of the weather, which is especially the case in February and March. It is quite surprising how successfully infants less than six months of age can be exposed to the rigorous climate of the winter months. In order to accomplish this without harm, the lower extremities should first be exposed, and at all times the feet and hands and trunk should be warm and well protected. In winter the exposures should be carried out at the warmest period of the day. In summer the morning hours should be chosen, so that the infant will not be overheated and fatigued. Heliotherapy is begun by exposure of the ankles and legs for about 15 minutes daily and is gradually increased to a period of an hour or more (Figs. 176, 177). Later the arms are similarly treated. As is well known, Rollier has employed this form of treatment in Switzerland with remarkable success for bone and glandular tuberculosis. It seems capable of being adopted, perhaps somewhat less successfully, for infants at sea level.

In addition to a specific treatment, the infant should have an appropriate diet containing egg yolk and green vegetables. It should be given plenty of fresh air. According to Finlay, massage is of definite value. If the child is large and heavy it should be discouraged from standing or walking, in order to prevent bowing of the legs. Beef juice and meat are of value in certain cases. Iron should be given where there is anemia. In some instances the saccharated carbonate of iron in doses of 2 gr. three times a day has seemed to bring about some calcification of the epiphysis, although the inorganic phosphate of the blood remained unaffected.

In cases of severe deformities due to rickets it may be necessary to result to orthopedic or surgical measures. A description of these procedures will be found in treatises or textbooks on these subjects. How-

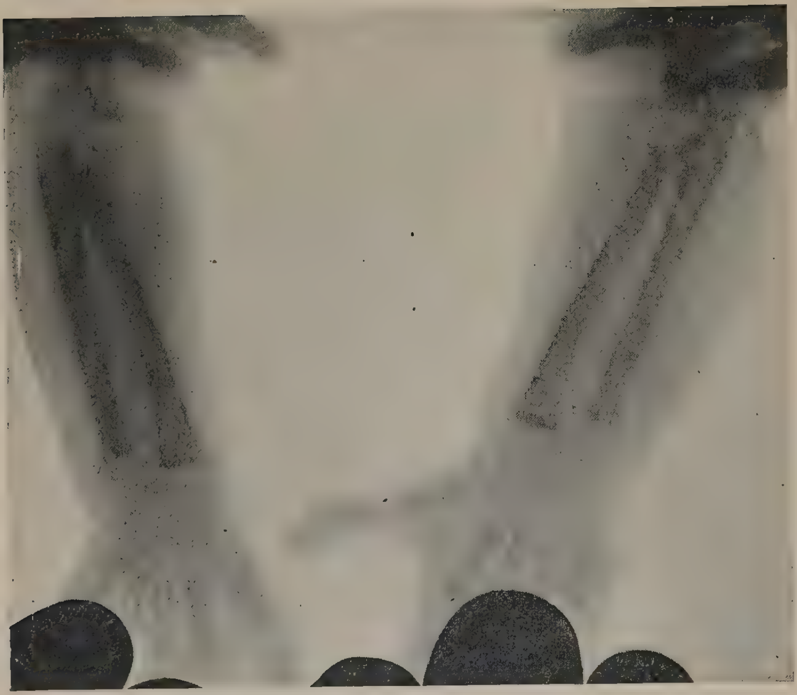

FIG. 176.—Moderate rickets. March 29, 1921.

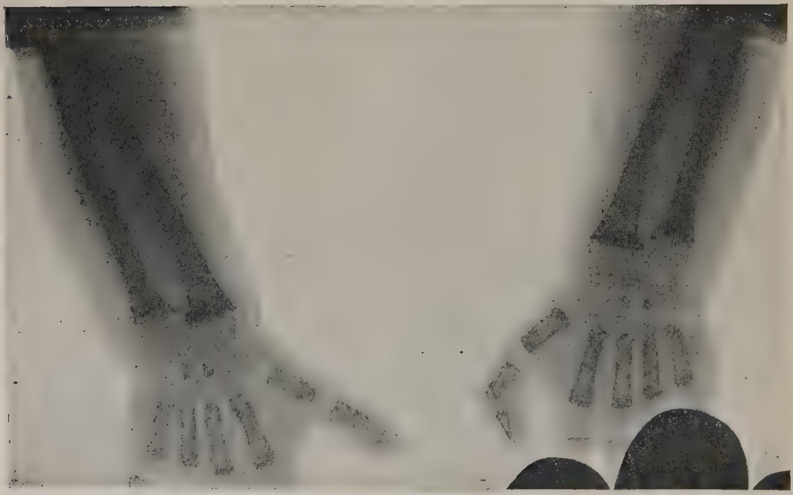

FIG. 177.—May 3, 1921. Same case as in Fig. 176. Sunlight treatment for five weeks, 15 to 30 minutes daily. Diet unchanged. Note marked calcification of epiphyses and development of two carpal centers.

ever, many cases of marked bowing of the legs correct themselves without treatment, as the children grow and develop.*

In the treatment of tetany, ultraviolet light and cod-liver oil are likewise specific and able to alleviate the symptoms and to increase the calcium content of the blood. The action, however, is slow as in rickets. In addition to these measures it is desirable to give calcium in order to quickly decrease the irritability of the nervous system and prevent convulsions. To bring about this result large doses must be given. As much as 50 or 60 gr. daily of the chlorid or 100 or 120 gr. of the lactate may be necessary in order to produce the desired effect. György,† who regards tetany as due to an alkalosis, and the calcium therapy as "an acid therapy" has recently reported good results from ammonium chlorid. This is given in doses of 75 to 100 gr. a day (in 10 per cent. solution) and is stated to control promptly the nervous manifestations.

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[*] We have striking examples of this among the negro children in New York City. Although rickets is almost universal, and was still more prevalent some years ago, the older children of school age have remarkably straight legs and bodies. In a public school in New York City where the attendance is almost entirely negro, bowed legs are not especially evident.

[†] György, P., Klin. Woch., Nr. 28 (1922), p. 1399.

CHAPTER XXXIII

THE CONSTITUTIONAL DIATHESES OF CHILDHOOD NEURO-ARTHRITIC DIATHESES; STATUS THYMOLYMPHATICUS; INFLAMMATORY EXUDATIVE DIATHESIS; SPASMOPHILIA AND TETANY

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The conception of a diathesis, or a constitutional predisposition, which induces in an organism some change from the normal is very old in medicine. That the exaggerated or altered reactions of an organism are due to its constitutional peculiarity is an idea which has survived the successive evolutions of pathologic thought, and today, as forcefully as ever, compels the attention of the clinician.

This is particularly apparent to the pediatrician, because the young organism, which is intrinsically less stable than the older organism, does not readily adapt itself to its environment under diathetic influences. The pediatrician is constantly reminded of the interaction between internal and external factors in the causation of disease, and the significance of individual predisposition in the incidence and course of health disturbances is continually before him. To interpret these relations in the terms of a diathesis is almost inevitable. The pediatrician has some advantage over the internist in this field, since the external conditions of existence are far more uniform in the infant than in the adult.

The time elapsing between birth and the first evidences of a predisposition to an abnormal reaction to disease processes is, in general, comparatively brief. In a word the infant gives the clinician the opportunity to witness at first hand the successive phases of an abnormal predisposition or constitutional diathesis. The fact is forced upon the observer that a congenital anomaly of the organism exists. It reacts abnormally to physiologic stimuli which in the average infant would evoke normal responses.

The pronounced influence of proper or improper feeding upon the pathogenesis or the amelioration of diathetic manifestations is the measure of the degree of metabolic disturbance. The fateful consequence of improper feeding is quickly established beyond a doubt. Nutrition or malnutrition is a secondary influence in the development of diathetic states. Food or feeding conditions under which the normal infant thrives may cause abnormal reactions in the diathetic child.

The following cases serve to illustrate some of these points:

Case I.—J. E., girl, aged 4 weeks, weighing 1800 gm., but seemingly well nourished, frequently regurgitated her food and was in almost constant distress between meals. A great deal of gas was expelled, and green stools filled with curds were passed frequently. There was extensive seborrhea of the scalp and slight intertrigo in the folds of the groins.

The mother and the father were both more than 30 years old. They were intelligent; but both were subjects of a constitutional predisposition. The father was a neurotic and was frequently troubled with migraine. Until the age of puberty, he had wet the bed. He feared the injection of serums because of the severe urticaria which developed subsequently.

The mother was of the plethoric type. She was much troubled with seborrhea of the scalp and eczema in the skin folds. There was considerable blepharitis marginalis. An urticaria developed after she had eaten certain foods, particularly those rich in fat. Her father had been troubled with eczema, and in later life with asthma.

Case II.—G. H., aged 5 months, was pale and fairly well nourished, the weight being 4860 gm.; but the tissue tone was poor. There was some enlargement of the cervical glands. The fontanel was open, and there was some craniotabes. The head was of a rachitic type, as was also the chest, which exhibited a rosary. There was some rachitic enlargement of the extremities.

The mother was unable to nurse the baby on account of inverted nipples. A wet nurse was nursing both this infant and her own. Each infant was given one breast, and both were nursed every four hours. The amount given at each feeding varied between 4 and 5 oz.

The wet nurse's infant was 8 weeks old, well-nourished, and had excellent muscle-tone and clear skin. It was practically free from "spitting" of the food; the stools were normal in appearance and were not frequent. The infant was seldom uncomfortable and slept during the greater part of the interval between feedings. The mother was a robust woman and in fine physical condition. She had always been free from skin lesions.

The disturbance in this case was corrected by limiting the quantity of breast milk given to 2 oz. at each feeding, and complementing it with 3 oz. of a full-strength buttermilk (Holland formula). A very small dose of atropin (1/2000 gr.) was given before each nursing. Lassar's paste was used locally. This method of feeding was continued until the third month. The food from that period until the sixth month consisted of a half-strength mixture of cow's milk, partly skimmed, and dextrimaltose. After the fifth month cereals, toast and vegetable pulp were added to the diet. The baby showed steady, normal gains in weight; was generally comfortable and has remained entirely free from exudative manifestations.

The chief complaint was of marked restlessness and disturbed sleep. The child was easily startled, held its breath when it cried, and frequently exhibited attacks of laryngospasm. It had several attacks of slight convulsions in the course of an acute nose and throat infection accompanied by fever. A definite Chvostek phenomenon was present. The electrical reactions were increased, the anodal opening contraction occurring at a half milliampere.

Both parents were robust and seemingly in good health. The mother had been told that she had convulsions during childhood. She exhibited a positive Chvostek phenomenon.

The infant was breast fed for three weeks and was then put on a half-and-half mixture of cow's milk and gruel with cane sugar, on account of the occurrence of breast abscess in the mother. This mixture had been modified to the proportions of two-thirds milk and one-third diluent. There had been marked constipation. On account of a pronounced laryngospasm gruel food alone was given for a period of forty-eight hours. Calcium bromid was ordered to be given by mouth. These combined measures reduced the electrical reactions to nearly normal. The infant was again put on breast milk and two doses of phosphorized cod liver oil were given daily. In the course of six weeks the tendency to laryngospasm had practically disappeared. The electrical reactions remained normal, and other latent phenomena of spasmophilia could hardly be elicited.

These diathetic cases are typical of those which, in more or less aggravated forms, constantly come under the care of the pediatrician. It is quite logical to assume that a congenital anomaly in such persons exists.

What the older writers designated as constitution, Martius describes as a supertype of physiologic reaction to external influences. The development of disease processes occurs according to the formula $\frac{P}{C}$ = pathogenic influence, measured by individual resistance. The total constitution is the sum of its partial or subconstitutions. There is no general resistance, only an individually variable degree of resistance against specific noxæ.

A diathesis may be defined as an individual, congenital condition, or predisposition, frequently transmitted, which manifests itself by abnormal reactions to physiologic stimuli. Living conditions or environment, which in the majority of the species produce no untoward effect, in the diathetic subject cause the development of manifestations of disease. The severer degrees of a diathetic state may be individually determined; but the milder types imperceptibly shade into the normal.

It is difficult to circumscribe the specific types. Few symptoms are exculsively peculiar to a single type; for instance, the galvanic hyperexcitability of spasmophilia. All degrees of diathetic peculiarity may develop at different times in one and the same individual. A review of the etiology of the diatheses in general suggests a close genetic relationship between the different types. Three principal types of diathesis are described and recognized as definite clinical pictures: (1) the status thymicolymphaticus, first described by A. Paltauf in 1889, and later in 1906, and more fully by Theodor Escherich; (2) the neuro-arthritic diathesis described in detail by Comby and Marfan in 1900, and known in earlier British and American literature as lithemia; (3) the inflammatory exudative diathesis, mentioned as early as 1872 by Thomas White, but more minutely defined and described by Czerny in 1905.

Spasmophilia, the disposition to rickets, and the nutritional disturbance classed by Pfaundler as heterodystrophy may also be classed as diatheses but are better considered as subtypes of the principal forms.

Certain symptoms or groups of diathetic symptoms exhibit characteristics as to period of occurrence, interrelationship, and localization which permit of their classification. (1) The manifestations of these diatheses occur upon slight provocation and sometimes are of seemingly spontaneous development. Frequently, they appear abruptly and rapidly and reach a high degree of intensity. Often they take the forms of eruptions, crises, or paroxysms, which may disappear as quickly as they come. (2) Characteristic symptoms are often accompanied by severe so-called reflex phenomena. Their catarrhal exhibitions are of the spasmodic order. (3) These symptoms occur particularly in infants who are in an excellent rather than a declining state of health. (4) They show a tendency to remain distinctly localized; e.g., the geographical type limited to the tongue; seborrhea confined to the scalp; eczema on the cheeks and chin; and urticaria on the body.

Pfaundler comes to the conclusion that the sum of the diatheses is divisible into a number of separate and peculiar predispositions. These are not subordinated to the group and occur independently one of another. They have no common origin, are of a pluricentric rather than a unicentric system. They consist of several associated individual predispositions frequently of synchronous manifestation.

As to the etiology of the diatheses, all observers are agreed that these conditions are of distinctly congenital origin. They are transmitted, moreover, in heteromorphous form. When the diathetic complexes are separated into their individual components, it is found that some of these are familial. Brothers and sisters, as well as their direct ancestors, are frequently affected by the same diatheses. Mothers are more frequently the transmitters than the fathers. Boys are more often affected than girls. The familial spread of these diathetic predispositions presents many characteristics of genuine hereditary transmission.

Martius would seek the fundamental cause of the transmitted diatheses in a congenital functional deficiency or defect of certain organs or tissues. According to him determinants with modified characteristics may occur, which are especially consequent in the pathogenesis of internal disease processes. Congenital anomalies of function and specific functional deficiencies may result in spite of apparently normal structural conditions.

Martius' theory is applicable to the hereditary transmission of infantile diatheses. In each instance it covers the apparently arbitrary mutation of the various distinct predispositions. Every special predisposition underlying general states, such as the lymphatic, the exudative, the vagotonic or neuropathic diatheses, may be referred to some one distinctly modified determinant. We may conceive of this modified determinant as impressed by the male germinal plasm, as permanently eliminated therefrom, or possibly as remaining latent for a generation.

The practical difficulty of applying Martius' theory lies in the fact that so many of the diatheses appear in associated forms. Martius attempted, however, to coördinate and arrange systematically the various conditions which are subject to hereditary transmission. Within the large main group of diathetic states, conditioned alike by qualitative alterations of their determinants, he recognized four subgroups based upon the occurrence and the exciting causes of their manifestations.

In the first group, the predisposition and its specific manifestations are fully formed at birth. In the second group, the manifestation first develops in the extra-uterine period and is associated with some developmental phase, e.g., puberty. In the third group, the manifestation throughout life remains conditioned by the incidence of certain external influences which lead to disturbances in the diathetic subject more readily than in individuals with normal disposition. In the organism as a whole certain tissues or individual organs are lacking in vital energy. These are, therefore, incapable of offering the proper resistance to these exogenous noxious influences. In the fourth subgroup conditions are similar, with the exception that manifestations of disorder or abnormal reactions occur to stimuli which ordinarily would fall within the class of normal excitants, such as occur ordinarily within the organism and are regarded as physiologic. The physiologic function itself becomes, as it were, the agent of the disturbance. The types of diatheses observed in children all come within groups 3 and 4.

If functional deficiencies in certain organs are the fundamental substrata of the diatheses, it should be possible by functional tests to demonstrate, even in the latent period, such deficiencies. The determination of food tolerance as suggested by Finkelstein is of value in this connection.

Czerny believes that the inflammatory exudative diathesis associated with a disturbance of the fat metabolism is an expression of a deficiency of the organism. The experimental studies of Birk and Meyer do not substantiate Czerny's views, but clinical observation shows that latent exudative symptoms invariably become very active under high fat feeding.

Czerny also suspects congenital defects in the tissues concerned in fluid retention. This involves disturbances in the mineral metabolism. In this instance, again, experimental data are at variance with the theory and with clinical observation. Bruch found the mineral metabolism practically normal in infants affected with marked indications of eczema and in subjects who would be classed as exquisitely exudative in the sense in which that term was used by Czerny.

In the neuro-arthritic diathesis, the French writers have assumed a tissue deficiency with regard to the purin metabolism, analogous to that associated with gout; but this assumption is not in accord with the experimental data so far produced. Göppert and Uffenheimer could not definitely establish any consistent abnormality in the purin metabolism of the neuro-arthritic subject. In a large series of carefully selected cases, both of the exudative type described by Czerny and of the neuro-arthritic type described by Comby and Marfan, and some of them rather extreme cases, I have found the figures for the blood nitrogen and all of its separate components practically those of the normal organism.

The marked reaction of lymphatic exudative types of children to vaccination or to the parenteral administration of foreign proteins is interpreted by Moro as an expression of heightened vasomotor excitability.

Some of the diatheses show characteristic latent phenomena, the discovery of which often gives valuable aid in establishing a diagnosis. The Erb, Trousseau, and Chvostek phenomena of the spasmophilic diathesis are examples of such latent phenomena. Hypereosinophilia is a valuable latent, although isolated, phenomenon of the inflammatory exudative diathesis. The peculiar general appearance characteristic of some of the diatheses, the so-called habitus, is often so pronounced that this or that type can be thus recognized at the outset.

While most of the diatheses are congenital it is undoubtedly possible to acquire a diathesis. The so-called biologic sensitizations resulting in anaphylaxis and brought about through the influence of certain antigens are in effect states of acquired diathesis. Serum disease, hay fever, and idiosyncrasies to certain foods or drugs probably belong in this group. The hypersensibility to cow's milk or to egg yolk in cases of periodic vomiting are typical examples.

The characteristics of the habitus of children affected with one, or with a combination of several, of these diatheses should be noted. Loosely arranged according to predominant features, three groups may be recognized:

I. The torpid, pudgy, lymphatic, anemic habitus. The skin of these children is generally pale or sallow. Their muscular tissue is soft, its tone dough-like. They are squat and pudgy. They look fairly massive, but are ponderous. All visible or palpable lymphatic glands are enlarged. Mouth-breathing is commonly present and is due to pronounced hypertrophy of the tonsils and to the presence of adenoid tissue. All the lymphatic structures of the nasopharyngeal ring tend to hyperplasia. In disposition these children are dull, indolent, torpid, and careless, exhibiting to a marked degree the characteristics of the phlegmatic temperament.

II. The neuro-arthritic habitus. Children of this type are inclined to be thin. The skin is transparent; the eyes are lustrous and the pupils are highly reactive. They are restless in disposition, impulsive, irritable, mentally very bright, and often precocious. In temperament they are extremely variable and inconstant. There is an inclination to affectation. Tasks are done in a flighty, careless manner. Emotions run to extremes, either in the way of exaltation or depression.

These children are generally bad companions and frequently prefer solitude. Habits are easily formed and, because of the adroit, secretive disposition of the child, are checked with difficulty. While mentally this type of child is acute and even brilliant, its acuity is confined usually to certain lines of activity. Comby has aptly called them "Les dégénérés supérieurs."

III. A third type has the plethoric or obese habitus. These infants or children show abnormal development and are generally very fat. Their color index is high. There is a marked tendency to skin eruptions and to profuse sweating. As such children grow older, the obese habitus may change to the neuro-arthritic type with all its attendant characteristics and with the complete disappearance of the earlier qualities.

The status thymicolymphaticus is generally found in children of Type I. The spasmophilic diathesis described by Thiemich, Finkelstein and von Pirquet is formed with equal frequency in this type; it may be either alone or associated with the status thymicolymphaticus. The neuro-arthritic diathesis is almost exclusively representative of the habitus of Type II.

The inflammatory exudative diathesis is most prevalent with the habitus of Type III. Frequently it is not present alone, but associated with one or more of the other diatheses. In fact the several diatheses seem rather closely related. There are those who believe that they are in reality not separate entities at all; but rather consist of a single condition capable of many-sided expression.

This impression tends to be confirmed when one carefully analyzes the descriptions given of the various forms by different observers. They either describe symptoms which are identical, or the different diatheses possess some characteristics which are essentially similar or are actually common to them all. This alternative adds confusion to the subject and makes it difficult to define individual pictures or properly to limit different diathetic states.

However, to determine whether we are dealing with a single constitutional dyscrasia capable of varied and manifold expression, or with several distinct diathetic types, is not so essential as is the recognition of the fact that a constitutional dyscrasia is of exceedingly common occurrence in infancy and childhood and has undoubtedly and unmistakably a profound relationship to the nutrition and development of the child and often accounts for reactions which are other than normal.

NEURO-ARTHRITIC DIATHESIS

In many of the older classical writings frequent reference is found to diatheses or constitutional dyscrasias. Thomas White (1788) speaks of a diathesis inflammation; Hufeland of a humoral exudation of scrofulous nature; Virchow speaks in similar terms of an inflammatory diathesis. Murchison at a later date (1877) first mentions the term lithemia. Sir Dyce Duckworth's views on gout, considering it a trophoneurosis, undoubtedly influenced the views of later English, American and French pediatricians. When they speak of a lithemic state or of neuro-arthritism, they have in mind a condition analogous to the gouty diathesis. The theories advanced by Sir Dyce Duckworth in explanation of the neuroses accompanying the gouty state are interesting and applicable to many of the nervous phenomena attending the diatheses of childhood. He believes that such a neurosis can be implanted or a tendency to it established and that it may be hereditary. Another possibility he suggests is the establishment of a secondary or induced neurosis due to some toxemia or blood degeneration. "The neuroses represent special conditions or special modes of evolution of nerve force. They are implanted in individuals as part of their intimate nature. They are truly as characteristic of the individual as are his features or other physiognomic traits. The nervous system or some definite part of it has a peculiar disposition or tendency toward the morbid evolution or manifestation of nerve function. It does not necessitate the existence of any gross or coarse disease, but it is a more or less abiding condition ready to manifest itself on suitable provocation."

The special characteristic of these neuroses is that they tend to be hereditary. Another feature is an element of distinct periodicity which attaches to them, with a paroxysmal tendency. There are, then, two fixed features, the one, the abiding element; the other, the proclivity to recurring outbreaks. In these neuroses a certain law of alternation or substitution prevails. We thus encounter certain affections in the parents and ancestors and others equally neurotic in the collateral relatives or descendants.

Rachford's description of lithemia or the lithemic state is the earliest reference in pediatric literature to a diathesis. Under this term, Rachford included all symptoms thought to be due to an excess of alloxuric bodies in the body media. Among such he mentions uric acid infarctions, accompanied by fever, paroxysmal crying and fretfulness, vulvovaginitis and urethritis; and in older children, by diurnal and nocturnal incontinence of the urine.

Toxic symptoms manifest themselves principally in the gastro-intestinal tract and in the nervous system. There may be gastric pain, nausea, and vomiting. All food may be rejected for four or five days. Temperature ranging between 101°F. and 105°F. may continue for a similar period. Prostration and loss of weight are pronounced. The familiar picture of cyclic vomiting is easily recognized.

Among nervous symptoms, those resembling an eclamptic state are most marked. Afebrile convulsions are not uncommon. Rachford speaks of migrainous or lithemic epilepsy. These attacks are commonly associated with migraine or with the already described gastro-enteric symptoms. The resemblance of this syndrome to the present conception of spasmophilia is quite suggestive. The lithemic organism is very subject to eczema and to such manifestations as angioneurotic edema. In the respiratory tract symptoms such as those of spasmodic asthma and bronchial tetany are often observed. Rachford also speaks of the peculiar mental make-up of the lithemic child, particularly of the tendency to mental precocity.

Between 1900 and 1902 Comby's publications on the neuro-arthritic diathesis appeared. In his description of this diathesis, or the condition of arthritism, as Comby calls it, all the views held by earlier English and American writers on lithemia are embodied. Comby, however, included many new symptoms and in an ingenious manner points out the relationship between this diathesis and these symptoms. He suggests also the widespread occurrence of these conditions, particularly in large urban centers.

As in part suggested by the term neuro-arthritic diathesis, Comby and Marfan advance the hypothesis that error in the purin metabolism underlies the development of all these symptoms. Experimental biochemical investigation has not borne out this hypothesis. The purin metabolism of these cases is generally quite normal, even in the extreme types. The tern arthritism is, therefore, a misnomer. This does not mean that the diathesis does not exist. On the contrary it is one of the most commonly observed conditions of infancy and childhood. The predisposition is distinctly hereditary. The neuro-arthritic infant is the offspring of neuro-arthritic antecedents.

STATUS THYMOLYMPHATICUS

The views and theories of Bouchard on constitutional diatheses had their influence over Comby and Marfan. This author admits of but two types of diathesis: (*a*) the scrofulous or lymphatic type; (*b*) the arthritic type. There are many features of Bouchard's scrofulous diathesis which suggest some relationship to tuberculosis. Comby and Marfan explain this relationship on the basis of the formula $\frac{\text{tuberculosis}}{\text{lymphatism}}$ = attenuated tuberculosis or scrofula. The lymphatic state is often apparent in infants born of ill or enfeebled parents. Such parents transmit to their offspring a cellular structure inferior alike in component quality and functional activity. This is held to explain the liability to nutritional disorders of such an organism. With a faulty heredity preponderant, such unhygienic influences, as inadequate air supply, lack of sunshine, and insufficient nourishment, play their mischievous part. Even during the early months of infancy, the lymphatic subject presents peculiar symptoms. Among these are marked pallor, flabby tissues, protuberant abdomen, slow torpid movements. A general adenopathy is commonly observed.

The lymphatic infant is sensitive not only to the tubercle bacillus, but also to other microörganisms, both pyogenic and saprophytic. This sensitivity subjects the child to constantly recurring infections of the skin and of the mucous membranes throughout the body. Coryzas and anginas are common in these cases. The tonsils tend to hypertrophy; adenoid tissue is redundant. The eyelids are prone to blepharitis marginalis. Chronic bronchitis is frequently in evidence at all seasons of the year. Balanitis or vulvovaginitis is often observed. The extensive involvement of the lymphoid tissues and the repetitional attack upon the mucous membranes tend to cause a characteristic facies. A dull, heavy expression, a large broadened nose, long straight eye-lashes suggest the scrofulous habitus of the older writers. That the skin of the lymphatic infant is as sensitive as the mucous membranes is indicated by the recurring tendency to impetigo, eczema, fissures, intertrigo, pityriasis, which often prove extremely rebellious to treatment. All the reactions are torpid. The recession of inflammatory processes is very slow. All lesions show a tendency to chronicity.

Schreiber points out the close analogy between the condition so pictured and the symptoms of the status thymicolymphaticus as described by Paltauf and Escherich. The rather constant findings of abnormal lymphatic structure in many cases of sudden death, the so-called mors thymica, attracted the attention of Paltauf and led him to suspect some form of dyscrasia in such subjects. Escherich recognized the imminence of this diathesis during the period of infancy and childhood, and minutely drew a clinical picture of the condition which emphasizes many of its most interesting pathologic features. He shows an hypertrophied thymus, an enlarged spleen and a hyperplasia of lymphatic structures in general as prominent anatomical features of this dyscrasia, to which he gave the name status thymicolymphaticus. Still other signs of this diathesis are seen in the marked pallor, the relaxed muscle tone and the abundance of adipose tissue. In the epiphyses there are definite evidences of rachitic changes. The lymph nodes, particularly in the cervical, retropharyngeal, axillary and mesenteric chains are enlarged. The scrofulous habitus and a tendency to prurigo and other forms of irritation of the skin are often associated with this diathesis.

Temperamentally such children are phlegmatic, and yet frequently they show certain acutely nervous phenomena, as laryngospasm and spasmic contractions of the muscles of the mouth and of the extremities. So frequent, in fact, is the association of laryngospasm with the lymphatic diathesis that Escherich believed that this status definitely predisposed its victim to this phenomenon and to other associated neuroses.

With the broader and more recent knowledge of the characteristics of what is known as spasmophilia it is now quite clear that many of the nervous phenomena which Escherich described were those of the spasmophilic state. It remains most probably that the two diatheses, the status thymicolymphaticus and the spasmophilic state, are very commonly associated.

INFLAMMATORY EXUDATIVE DIATHESIS

In 1905 Czerny first published the description of a constitutional status for which he suggested the name inflammatory exudative diathesis. Under this term Czerny attempts to combine in a single entity a number of disease complexes previously treated as individual. The entire clinical complex of the status thymicolymphaticus, for example, Czerny considers as a form only of the inflammatory exudative diathesis.

At present it is difficult to say whether the major diathesis which Czerny describes is entitled to the place he accords to it as a grand entity or whether it is merely an admirable new name for groups of conditions previously described under distinctive terms. All the principal symptoms of such diatheses as the status thymicolymphaticus, the lithemic, and the neuro-arthritic predispositions reappear in some form or other in Czerny's picture of the symptomatology of the inflammatory exudative diathesis; and to the older complexes he adds many new features. The older conception of scrofulosis is presented in an entirely new light and is considered as simply a part of the symptom complex of the major diathesis.

Czerny defines the exudative diathesis as a congenital anomaly of the human organism which generally involves all the members of a family, but may express itself in a varying degree in its several individual members. The congenital quality of the diathesis is often demonstrable. Transmission of the diathetic predisposition seems to be less positive if the dyscrasia runs on the paternal side; more definite if it exists on the maternal side, and practically assured if it obtains on both sides of the family.

New-born infants are distinctly dissimilar, and particularly so in their tissue reactions. Under similar conditions of feeding one infant thrives splendidly; a second becomes anemic; a third, rachitic, and a fourth develops nervous phenomena. Czerny advances the explanation that these various clinical phenomena are dependent on the fact that infants are born with the chemical organization of their tissues in differing stages of development. Several constitutional anomalies in the chemistry of tissues may be combined. As a result, various types of diathesis may simultaneously appear in the same infant.

The manifestations of the inflammatory exudative diathesis depend essentially on the sort of food the infant is receiving. The symptoms manifest themselves mainly on the skin, in the mucous membranes, and in the lymphoid tissues. The skin conditions include seborrhea of the scalp, milk crust on the face, strophulus, and all the various forms of eczema on the body surfaces and of intertrigo in the skin folds. In the mucous membranes one of the earliest manifestations is the peculiar irregular desquamation of the tongue surface, which Czerny calls the *lingua geographica*. Most of the mucosæ show tendencies to periodic, recurrent or chronic catarrhal attacks; in the way of conjunctivitis, rhinitis, nasopharyngitis; and, in the respiratory tract, laryngitis, pseudocroup, asthma, and relapsing bronchial catarrhs. Certain types of intestinal catarrh are common, and inflammatory conditions of the genitalia, as balanitis and vulvovaginitis, frequently appear in such infants. The lymphoid tissues show a tendency to hyperplasia. The tonsils are liable to be large. There is a general enlargement of lymph nodes and of such organs as the spleen and the thymus. On the other hand, the manifestations of the inflammatory exudative diathesis often remain latent for variable periods of time.

The intimate interrelationship between this condition and the nutrition of the child is very apparent. Certain definite reactions to unbalanced feeding often furnish the clue to a latent diathesis. The potentially exudative infant is slow to regain its initial weight loss, notwithstanding an adequate quantity and suitable quality of food. Whenever the diathesis is latent some symptoms will sooner or later appear.

Maternal nursing often seems to disagree with the child. The quality of the milk is questioned and may be considered the chief causative factor in the disturbance, and weaning is advocated. To conclude that the mother's milk is at fault is manifestly an error, as may be proved by the following familiar observation. In institutions a single wet nurse will often nurse several babies, including her own. Her own infant may show symptoms of inflammatory exudative diathesis while one or all of the others show none. Again, the very reverse may be true. Her own child may remain free from symptoms, whereas others under her feeding may exhibit them in more or less marked measure. Observations like these would seem to emphasize the hereditary nature of the diathesis. The nutriment given seemingly causes the appearance of exudative phenomena only in the organism hereditarily predisposed.

Improper feeding, however, will favor the outbreak of symptoms of the inflammatory exudative diathesis, while suitable food, suitably fed, will control the outbreak of further symptoms or will abate such as are already manifest. Czerny believes that the connective and adipose tissues have characteristics differing from the normal in those who are affected with an hereditary inflammatory exudative diathesis. These peculiar qualities persist into later life. Any error in diet will, even in adolescent or adult years, excite symptoms of disturbance similar to those which appear during the period of infancy and early childhood.

Injury to the epithelial layers of the skin in the normal infant causes a slight exudation with rapid coagulation of the exudate. In the exudative organism the behavior is altogether different, especially if the infant is of the florid, fat type. The least irritation of the denuded area delays the coagulation of the lymph for a long time. The result is considerable persistent oozing or weeping of the injured surface with the subsequent formation of crusts. The mucous membranes behave in a similar manner. It was this peculiar behavior of the skin and the mucosæ which caused Czerny to name this predisposition the inflammatory exudative diathesis.

Much clinical evidence points to the fat component of the food as the chief factor in provoking the appearance of symptoms. It is more true probably that any type of food or of feeding which favors rapid gains in weight seems to excite the tendency of the skin and the mucous membranes to the exhibition of inflammatory exudative phenomena; while the reverse holds true of the food or the type of feeding which aims at reductions of weight.

The disappearance of exudative symptoms during periods of protracted illness, attended by pronounced weight loss, and their reappearance during convalescence, especially when gains in weight are rapid, clinically substantiates the theory that the most intimate connection exists between the nutritive status of the child and the development of the predisposition to the exudative diathesis.

The lower fatty acids formed in the intestinal tract in the process of digestion are much more irritative to the infant affected with this diathesis than to the normal child. This is true even of the breast-fed infant. This helps to explain the occurrence of green stools, with abundant mucus, and slight recurring tendency to diarrhea so frequently observed in the exudative infant whether naturally or artificially fed. Although the diet in the exudative type of child is the indirect influence in provoking the exhibition of symptoms, it serves, after all, only to prepare a favorable soil for their development. Their actual outbreak is almost always referable to some external insult or irritation. The skin presents abundant opportunities for the infliction of these insults, a fact which suffices to account for the exudative predilection for this tissue. The tendency is not quite so great with the mucous membranes, especially the respiratory tract. Smoke and dust are variable factors in the atmosphere. Many localities are entirely free from these pollutions. An infant so favorably situated atmospherically, as for example at the seashore, in mountainous regions, or in the country, may never show respiratory symptoms although it may have a typical array of exudative symptoms in the skin. The inflammatory exudative state doubtless causes greater sensitivity of the respiratory passages to infection. It is this fact, together with the decomposition of the hypersecretions so formed, which is responsible for the ready rises of temperature so commonly observed in the diathetically disposed subject. An obscure temperature in an infant should always arouse suspicion that some exhibit of an exudative diathesis may be the underlying cause.

In the older child the disturbance may manifest itself in more chronic forms, particularly if no attention has been paid to proper feeding. The symptoms attending these attacks are fever, anorexia, coated tongue, offensive breath, and swelling of the posterior cervical glands. Anorexia is usually the most pronounced and the most obstinate evidence of the disturbance. The resulting undernutrition affords the reason for bringing the child to the physician. Careful investigation reveals the underlying exudative diathesis and the specific characteristic affection of the upper respiratory tract.

Czerny believes that the status thymicolymphaticus described by Paltauf and Escherich is identical with the inflammatory exudative diathesis, plus the particular feature of enlargement of many or all of the lymph nodes. He points to the intimate relation between the pathologic adipositas and this hypertrophy of the lymphoid tissues, a relation which is peculiar to the organism affected with an exudative diathesis. Czerny is influenced in his judgment that these two diatheses are identical by the fact that the same diet which will cause pronounced symptoms of exudative diathesis to disappear will also prevent the development of most of the distinctive symptoms of the status thymicolymphaticus or will cause those that are already present to recede and disappear. The lymphoid tissues, seemingly influenced by suitable feeding, are the thymus, the spleen, the tonsils, the lymph nodes of the intestinal tract, and to a lesser degree those of other regions. Czerny also calls attention to the interesting fact that the food most free from purins, namely the fats, will produce these unfavorable results, whereas a purin-rich diet will not produce the same effects.

Enlargement of the tonsils is observed chiefly in the second and third years. This is also the period within which a large thymus or spleen is usually found. The event of *mors thymica* occurs most frequently within these years. Czerny points out an interesting fact with reference to the general enlargement of the lymphatics. Feeding disturbances, largely resulting from the presence of the exudative diathesis, may keep the infant under weight during the first year. Efforts to secure gains in weight meet a better response during the second and third years; but simultaneously with these gains, and particularly if they are induced rapidly by forced feeding, a general enlargement of the lymphoid tissues is observed.

The clinical picture of an exudative diathesis is very complex, and the symptoms it presents may assume many differing forms. While it is always definitely hereditary it does not follow that identical disturbances will affect all the members of an affected family. Some may not show any manifestations at all; others exhibit severe skin symptoms; and still another member of the family may develop asthma or other affections of the respiratory tract. The tendency to skin lesions seems to abate as the child grows older, owing, in all probability, to the greater resistance to infection that the skin acquires.

Czerny justified his inclusion of all these manifestations and symptom complexes under the one comprehensive conception of inflammatory exudative diathesis on the ground that all of them may be influenced by suitable dietetic therapy. An inflammatory exudative diathesis cannot be cured by this dietetic treatment, but all of its symptoms may be held latent by this means to such a degree that they will be no longer troublesome and will not bar the development of the child. Definitely to achieve this result it is necessary not only to follow a correct dietetic therapy, but also to reduce to a minimum such external insults or irritations as may be caused, for instance, by improper clothing or by exposure to such mischievous atmospheric influences as dust and smoke. Such infections as measles and tuberculosis and such procedures as vaccination will often aggravate an existing exudative diathesis.

SPASMOPHILIA AND TETANY

Another form of diathesis which exhibits as its chief symptom complex certain nervous phenomena is the so-called spasmophilic diathesis. As is true of all the diatheses previously discussed, many of the symptoms and underlying pathologic conditions of spasmophilia have long been recognized.

There are good descriptions of infantile tetany in the earlier literature. The thought has occurred to many of its observers that they had to deal with some form of constitutional predisposition. Cheadle, many years before the investigations of the present day had thrown light on this important subject, expressed, to all intents and purposes, the modern conception of infantile tetany or spasmophilia in the following words: "In the vast majority of cases there exists as a predisposing cause a special constitutional state, in which the nervous system is in an extreme state of hyperexcitability or mobility, a state of erethism, as it is called, and in this state slight causes of irritation which would be innocuous in a condition of stability set up the muscular motor spasms of laryngismus, tetany, or general convulsions." The intimate relation, however, of this diathesis to the metabolism and nutrition of the organism was not recognized.

Under the term spasmophilic diathesis a constitutional anomaly is now understood which is recognized as a result of measurable or mechanical means of determining the excitability of the nervous system. This anomaly exhibits itself in a pathologic predisposition to certain partial or general clonic and tonic spasms or convulsions. This diathesis is very commonly associated with other predispositions and especially with the status thymicolymphaticus or the inflammatory exudative diathesis. It is also frequently observed in connection with rachitis. This relation is so striking that observers (Kassowitz and, more recently, Brown) look upon spasmophilia as part of the symptom-complex of rachitis. This view is not sustained, however, by experimental proof.

Spasmophilia is seldom seen before the fourth month and rarely occurs after the third year. There appears to be a definite seasonal element in its manifestations. All its symptoms are exaggerated during the fall and winter months and abate or almost entirely subside during the summer.

Of the probable causes underlying the development of this diathesis, many theories have been advanced. Some authors look upon it as a phase of the expression of the status thymicolymphaticus since the two conditions are so frequently associated. While it is true that spasmophilia is often seen in the pale, torpid, lymphatic type of child, it is with almost equal frequency observed in the thin, neurotic type, —in one affected, probably, with the neuro-arthritic form of diathesis. Deficiency of parathyroid function, with resulting abnormal calcium metabolism, has been thought to be a possible cause. Escherich has observed hemorrhages in these organs at necropsy in infants dying suddenly during a spasmophilic attack. Other observers (Grosser and Baetke) state that this is not a constant finding; and Auerbach found that it not infrequently occurred in infants who during life had been entirely free from any spasmophilic manifestations. Noel Patton's theory that the spasmophilic state and its symptoms depend on an intoxication by guanidin and methyl guanidin is so far not supported by sufficient experimental proof.

The spasmophilic diathesis shows the most intimate dependence upon the metabolism and nutritive status of the child. Through the quality and the quantity of the food it is possible to bring out spasmophilic symptoms in those that are predisposed or to cause manifest symptoms to subside. The breast fed infant is practically free from this diathesis. It is only in isolated cases that latent or manifest spasmophilia is observed among breast fed infants. Its incidence will be found to be distinctly familial. The artificially fed infants, and especially those fed exclusively upon cow's milk, furnish the largest number of active cases. The contrast between natural and artificial feeding in their influence over this diathesis is very striking. The relationship is so definite that it is actually possible to bring out a latent spasmophilia by simply changing from breast feeding to artificial feeding; and, vice versa, to cause the recession of many of the spasmophilic phenomena by merely discontinuing breast feeding.

Considerable evidence points to an abnormal mineral metabolism in this diathesis. Sabbatani's findings that calcium applied to the brain would diminish its irritability to electrical stimulation, and that substances causing a precipitation of calcium would produce an increased irritability first attracted the attention of investigators to this element and to the possible rôle it could play in conditions causing hyperexcitability of the nervous system. At Czerny's suggestion, Quest determined the calcium content of the brain of children dying from spasmophilia. He found the calcium content of the tissues in these cases distinctly lower than that of normal brains. Similar findings were reported by Cohn and by Aschenheim. These interesting findings stimulated considerable investigation. A general deficiency in the spasmophilic organism of the elements calcium and, possibly, magnesium is suspected. There is a retention of the alkali phosphates particularly potassium and sodium with a diminution of the calcium salts. Stölzner's theory that the condition is the reverse of a deficiency and that spasmophilia is rather due to an excessive accumulation of these elements has been disproved and is not accepted. Rosenstern found that the administration of calcium chlorid solutions (3 per cent.) would reduce heightened electrical reactions; and von Pirquet, Bogen, and Riesel observed similar effects with other calcium preparations. Behrend found the same thing to be true when magnesium sulphate solutions were given subcutaneously. The excellent researches of Howland and Marriott seem to show convincingly that a reduction of the calcium content of the blood is the prime cause in the production of tetany. Some unknown factor causes this loss. When such a reduction reaches between 6.0 to 7.0 mg. of calcium per hundred c.c. of blood serum, frank evidences of tetany appear.

(Note.—Normal Ca content of blood serum they find to be between 10 and 11 mg. per hundred c.c.)

The symptoms continue to be active as long as the calcium content remains low, and they become latent or disappear altogether when it rises. They also find consistently that the administration of calcium chlorid by mouth will cause an increase in the calcium content of the blood serum. Coincidently with its rise the spasmophilic symptoms disappear and do not recur so long as its use is continued. These authors also record the interesting finding that in convulsive disorders other than tetany there is no reduction in the calcium content of the serum.

The symptoms of spasmophilia may be divided into two groups: the manifest and the latent. The chief symptom of the manifest form is the carpopedal spasm. This is a form of tonic spasm often associated with paresthesias and involving one or all of the extremities. The upper extremities are almost invariably involved. The hands show the peculiar so-called "obstetrical position." The arms are often held against the chest. The lower extremities are not always involved, but if they are, they will show a flexion at the knee-joint, while the feet are held in the position of equinovarus. The spasms may continue for hours or even days. There is often marked hyperesthesia and in some cases edema of the dorsal surfaces of both hands and feet.

Another manifest symptom is the so-called eclamptic state. It is characterized by a type of convulsion peculiar to the spasmophilic diathesis. It offers a slight resemblance to the epileptic type of convulsion, and consequently the eclamptic state is frequently mistaken for that disease. There are, however, distinct differences between the two. The attack begins with a tonic form of convulsion followed almost immediately by a clonic state. The spasm generally starts in the muscles of the eyes and the face and then rapidly extends to the extremities. The clonic twitchings may be very severe. The breathing is quite labored and is accompanied by marked cyanosis. After a few moments general relaxation sets in, and consciousness returns almost immediately, the attack differing in this respect very decidedly from an epileptic seizure. The child may appear entirely normal after an attack.

The latent stage of tetany is the one most commonly seen. There are certain definite symptoms by which it is characterized and which may be individually or collectively present. A most pronounced symptom and one which usually manifests itself earlier than any other is that of laryngospasm. This is observed oftentimes as early as the fourth month. In mild form it manifests itself as a peculiar crowing inspiratory sound, often amusing to parent or attendant and looked upon as a mark of precocity in the infant. In the more severe form it may prove a formidable symptom, causing marked dyspnea, deep cyanosis, an anxious terrified expression, combined with convulsive inspiratory efforts. This picture persists for a few moments and is then followed by relaxation and gradual return to normal respiration. At times general convulsions may supervene upon a severe attack of laryngospasm. Any slight disturbance, such as crying, gaping or marked distention of the stomach, may precipitate an attack. It is always dangerous to make throat inspections in cases showing laryngospasm. Sudden death is not at all uncommon during an attack.

Other signs of latent spasmophilia are the Trousseau, the Chvostek, and the Erb phenomena. The Trousseau phenomenon is elicited by firmly compressing the arm in the bicipital sulcus, using either the hand or a rubber band. The constriction must be sufficient to cause a blanching of the distal part. When this occurs the child's hand will assume the tetany position peculiar to that of the spasmophilic infant. This phenomenon is not constantly present. The Chvostek phenomenon is also called the facial phenomenon. It is elicited by briskly tapping the cheek over the superficial course of the facial nerve. In the spasmophilic subject this procedure is instantly followed by lightning-like contractions extending to all parts of the face controlled by the facial nerve. The Weiss phenomenon is similar to the Chvostek and consists in a contraction of the orbital muscles. It is elicited by striking the nerve along its course in the temporal region. The facial phenomenon is the most sensitive of the latent spasmophilic phenomena. It is very constant, occurs early, and is often the only sign present which suggests this diathesis.

The Erb phenomenon represents the hyperexcitability of peripheral nerves as determined by the use of the galvanic current. The contractions which occur at the opening and closing of the circuit at various degrees of amperage have been formulated into a table by Thiemich. Von Pirquet has projected a schematic arrangement which permits of convenient charting of the various values. The kathodal opening and closing contractions and the anodal opening and closing contractions are the indices of functional response.

Thiemich and Mann have laid down the general rule that a kathodal opening contraction occurring within 5 milliamperes is pathognomonic of an exquisite latent state of spasmophilia. Von Pirquet, after extensive determinations, considers the anodal opening contraction as the sensitive index. An anodal opening contraction occurring at one-half, 1, or even 2 milliamperes definitely indicates the presence of a latent spasmophilic diathesis. Von Pirquet's view is probably the more correct and is sustained by practical experience. It is very difficult to obtain a kathodal opening contraction under 5 milliamperes in any case of spasmophilia even if all other symptoms are pronounced. The anodal opening contraction, however, will occur in mild or even latent cases on a comparatively low amperage and is a very constant reaction.

The galvanic hyperexcitability is without doubt the most sensitive index of spasmophilia. It is considered so pathognomonic of this diathesis that Fleiner, Thiemich and many other investigators consider any convulsive state in which heightened galvanic reactions are absent as not properly an expression of tetany or of the spasmophilic state and probably nonfunctional in character.

The peculiar muscular hyertonicity observed in certain cases of carbohydrate overfeeding, first described by Gregor, shows heightened galvanic reactions. It is classed as a form of tetany related to spasmophilia. The spasmophilic state is a functional condition and is not dependent upon the relative development of the peripheral and central nervous systems. This is proved by the fact that both galvanic hyperexcitability and other latent phenomena of spasmophilia are variable under influences of proper or improper methods of feeding. It is shown, further, by the fact that a spasmophilic state, uninfluenced by therapeutic measures, may continue for months with varying degrees of intensity. The influence of food and of the method of feeding upon the spasmophilic diathesis is an established fact. The observations of Gregor determined this with exactness. All the clinical manifestations of spasmophilia and, in particular, the galvanic hyperexcitability are promptly affected by the type of feeding employed. The breast-fed infant seldom manifests this diathesis. In the artificially fed child a return to breast feeding is often the only therapeutic measure necessary to effect a cure or a recession of symptoms. Gregor found that complete withdrawal of all food, combined with a liberal supply of water, would cause an extreme degree of hyperexcitability to disappear almost entirely within twenty-four to forty-eight hours. If breast feeding immediately succeeded this starvation period, the galvanic reactions would keep within the normal. A return to artificial feeding would promptly cause the reappearance of spasmophilic symptoms.

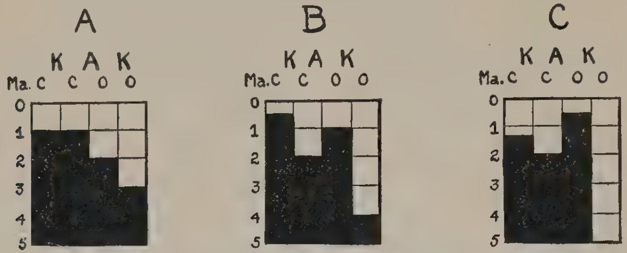

Fig. 178.—Von Pirquet's schematic chart; each space represents a milliampere; the letters above each column indicates the kathodal or anodal opening or closing contraction: *A* and *B*, charts of cases with pronounced electrical reactions; *C*, chart of latent case of spasmophilia with a high anodal opening contraction.

Finkelstein groups infants affected with the spasmophilic diathesis into two general classes: (1) the obese, overfed infant in whom the spasmophilic phenomena subside quickly if the food intake is curtailed; and (2) the thin undernourished child, subject to chronic gastro-intestinal disturbance, in whom a reduction of food supply will have no influence on the spasmophilic symptoms. Most of the spasmophilic subjects show more or less evidence of rachitis. The explanation of this peculiar coincidence is probably found in a metabolism common to both conditions.

The seasonal effect upon the spasmophilic child is interesting. The maximal effect is observed during the months of February and March. No adequate explanation of this influence has been offered. It has apparently no connection with the coincidence of rachitis. The spasmophilic diathesis is intensely familial and hereditary. Maternal transmission is much more definitely established than is the paternal. Regarding the fate of the spasmophilic subject in later life, there are observations (Birk and Thiemich) which seem to show that there is quite a definite relationship between spasmophilia in infancy and childhood and neuropathic phenomena in adult years. The development of the spasmophilic subject, with respect to mentality and nerve function, may not be quite normal. It is probable that the spasmophilic subject has, *a priori*, a nervous system which is structurally inferior. The deficiencies which appear in later life are traceable to this, rather than to any immediate effects of spasmophilic disturbances, however profound. It is a curious fact that epilepsy is practically never discovered in the antecedents of a spasmophilic subject.

The age period at which most spasmophilic phenomena occur lies between the third or fourth month of infancy and the close of the third year of childhood. This is, generally speaking, the period during which epileptic seizures do not commonly occur. Some of the manifestations of spasmophilia, especially the latent symptoms, such as the facial phenomenon, may persist for many years, and even into adult life. For treatment see page 984.

It is not difficult to understand, from the preceding descriptions of their several forms, that the subject of the diatheses is a rather complicated one. There is still much that is vague and theoretical in our conceptions, which must be tested out by more complete investigation and proved by sound experimental data. Many of the clinical phenomena they present are difficult to explain; the several types are difficult to define, and their separate identities difficult to establish.

The methods of treatment of the first three types of diathesis described are in many of their essentials strikingly similar, while that of spasmophilia is of a somewhat different order and may be separately considered.

From the initial appearance of the subject of the constitutional diatheses in medical literature, three essential elements have been recognized in their therapy: diet, hygiene, with particular reference to climate and environment, and hydrotherapy. The precepts of Comby and Czerny bearing upon the application of these essentials have found wide acceptance and if carefully and conscientiously carried out will give satisfactory results.

The subjects of the diathesis are generally of one of two types: the obese, torpid individuals, or the thin neurotic subjects. The former, in point of numbers, predominate greatly over the latter. The general indication is, therefore, for severe retrenchment in the dietary.

This is achieved by suitable selection of the food and by the avoidance of frequent feeding. In infants long intervals must intervene between feedings (the four-hour schedule), and the older child must be given only three meals a day, with the denial of all food between these meals. The obese, torpid type reacts unfavorably to overfeeding. The thin, neurotic type, on the contrary, is often benefited by gains in weight. The infant affected with constitutional diathesis should be breast fed. The quantity given at a feeding must be carefully controlled, and an interval of four hours between feedings must be strictly adhered to. If artificial feeding must be resorted to, the milk mixtures with reduced fat content give the best results. It is advisable to give well-cooked solid food in the form of cereals, vegetable purees, toasted bread, and stewed fruits as early as the sixth month. These things are combined with the breast feeding, if it can be maintained, or with the milk mixtures and are best given at the feedings which occur in the day-time.

A mass of clinical evidence seems to show that it is desirable to keep the milk supply as low as possible, even though, for a time, there is only a moderate gain in weight, or the weight remains stationary. As soon as possible the weight may be increased again by adding farinaceous and vegetable foods. The older child does well without milk and should be restricted in his fat intake. A diet consisting mainly of vegetables gives the best results in these diathetic states. Lean meats, and particularly fowl, may be permitted. There is some division of opinion concerning the use of eggs. Comby recommends their use. Czerny considers them harmful, both on accout of the anorexia they are liable to produce and the unfavorable effect they have on acute exudative skin symptoms. The organism affected with a constitutional diathesis seems to be more allergic to this form of food than is the normal subject. Schloss was able to show this in his valuable work on food allergy. Richet also mentions similar findings. In some experimental work on anaphylaxis it was found that patients suffering from periodic vomiting are sensitized to egg yolk. Comby recommends the use of alkalis and alkaline waters. They seem to be beneficial in the event of periodic vomiting.

Skin manifestations, such as the eczemata, intertrigo and urticaria, are always favorably influenced by a strict dietetic regimen. This with suitable local treatment (Fox's paste, Lassar's paste, resorcinol ointment, U. S. P. etc.) will, in most cases, effect control of the more troublesome symptoms. The itching is more intense in the thin neurotic type of child than in the plethoric obese type.

The psychic environment of the neurotic child must be regulated. This is, in fact, an important phase of treatment, particularly in the neuro-arthritic type of constitutional diathesis. The multiform nervous manifestations of these children, their erratic temperament, their disinclination for food, amounting at times to complete anorexia, are often met or favorably influenced only by a radical and complete change in their psychic surroundings. The anxious, intellectual parent, typical antecedent of such a child, presents the first and often the most pressing problem. If it can possibly be done, the child should be removed from the immediate home environment and should be placed among strangers. This measure is always attended with success. Sometimes it is well to place such a child with a group of companions, particularly during meal hours. The example of seeing others eat with a good will and healthy appetite is impressive and the example is often readily followed. Such a scheme may be easily carried out under institutional care, but it is also possible under private conditions, as is instanced in the work now being carried on by Emerson in Boston.

The climatic factor is a very important one. It is an old observation that the scrofulous subject will improve rapidly only if removed from the congested urban centers and placed in the country under favorable conditions of pure air and abundant sunshine. The smoke and dust-laden atmosphere of the great city is harmful, particularly so in view of the tendency to catarrh of the respiratory tract which many of these subjects exhibit, particularly those suffering from inflammatory exudative conditions. The dry, clean air of mountain plateaus or of the seashore, especially in the warmer latitudes, is ideal. The conditions secured should be such that the child may be out of doors most of the time and may wear a minimum amount of clothing. Under a regimen of this kind most of the catarrhal symptoms of the mucous membranes and many of the skin disturbances will markedly improve or will entirely disappear. The psychic state of the patient shares in the improvement.

Possible sources of focal infection must receive careful attention. Prophylactic dentistry and orthodontia are invaluable aids. The physician is only too often insufficiently alive to the usefulness of these corrective measures.

Hypertrophy of the tonsils and the formation of adenoid tissue are characteristics of the constitutional diatheses and predispose the child to all the infirmities resulting from such overgrowths. Whether radical, surgical removal of these structures is the best procedure, or whether one should rely on the conservative and slower methods, including dietary regulations, change of climate, etc., is a much debated question to be decided in each individual case. If the child is compelled to remain where he may easily become infected and where inflammation of these tissues is readily possible and frequently occurs, it is probably the best practice to remove the tonsils and adenoids completely. The benefits derived from the operation are often striking. A baffling anorexia disappears entirely; asthmatic attacks are lessened in frequency and in severity or may disappear altogether. The same thing is often true of attacks of periodic vomiting. The general physical development improves coincidently in a notable degree.

There is little need of medicinal treatment in constitutional diathesis. Cod liver oil, combined with creosote carbonate, is useful, as is some such preparation as the carbonate of iron or a modified Blaud's pill.

Treatment of Spasmophilia or Tetany.—The treatment of the spasmophilic diathesis is a somewhat different matter from that of the other forms. The infant should receive only breast milk. If this cannot be obtained at all, the milk mixture used in substitution should be as fat-free as possible. Semisolid food, as cereals, vegetables and toasted bread, should be given. For the older child a strictly vegetarian diet, with practically complete elimination of cow's milk and most of the fats, gives the best results. It is well, in any case, to avoid red meats, but other kinds may be given.

The administration of a 3 per cent. solution of chemically pure calcium chlorid will cause pronounced spasmophilic symptoms to subside promptly, and the more surely if a short complete starvation period of thirty-six to forty-eight hours precedes its use. If breast milk is then given and the use of the calcium continued for a long time, in the form either of the chlorid or the bromid, the symptoms usually remain completely under control. Phosphorized codliver oil (Ol. Phosphorat., 1.0 c.c. Ol. Morrh. 100.0) is a valuable remedy in spasmophilia and should be given over a long period of time.

If convulsions supervene, chloral hydrate should be used freely. The effect of this drug is rapid and safe. After the attacks have subsided, calcium bromid should be continued and the dietetic regimen reëstablished. Such measures will keep most of the spasmophilic phenomena under control and, if continued sufficiently long, will cause them to subside and to remain entirely latent.

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ACRODYNIA

CHAPTER XXXIV

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Synonyms.—Among the great number the most common are: Epidemic Erythema, Acrodynie, Krämpfsucht, Kornstaube, Kriebelkheit, Morbus Cerealis, Epidemicus Malignus, Podalgia, Ignipedites, Pink Disease, Erythroedema.

History.—It is not definitely known where this disease originated or even how long it has existed in Europe. The first authentic account of its presence there was in the latter part of the 16th century.

From then until the present time it has been present in some country of Europe either in epidemic form or as an occasional sporadic outbreak.

Within the past four or five years since interest in the subject has been aroused by reports from America and Australia, the disease has been described in practically every country in Europe.

Most of the epidemics were of a mild type until the latter part of the 18th century when the disease visited several sections of Germany in rather severe epidemic form. The symptoms of the disease vary with different epidemics and different localities. This naturally created confusion as to its identity and it proved difficult to determine whether one or several disease types were being encountered. These differences of opinion found expression in many titles under which the disease was described. That all the authors were describing the same disease with characteristic variations is no longer a matter of serious doubt.

It was not until the great French epidemic which lasted from June 1828, until the winter of 1830 in which from forty to fifty thousand people were attacked, did the disease receive the attention at the hands of medical writers its importance would seem to justify. This epidemic was described by many of the most distinguished medical men of France. Among the most notable were Cayol, Alibert, Dance, Kayer, Delmas, Chomel, Genest, Valliex and Dezeimeris. The recorded clinical observations made by these men, their attention to the minutest detail both as to environment and circumstance has so illuminated the subject that whatever doubt might have existed as to whether this was a disease entity or a mere symptom-complex was definitely settled in favor of the former view.

The writers mentioned above adopted various titles in describing the epidemic; such for instance as "The prevailing Epidemic,"

Epidemic Erythema." For the name acrodynie (English—acrodynia) which has been generally adopted by those writing upon the subject we are indebted to Chardons, fils, who in 1830 published an article in Revue Medicale franc. et etrang. entitled "Del'acrodynie, ou'epidemie qui a regne a Paris et dans les environs depuis l'annee 1828."

It appears that even at this time acrodynia was not confined to Europe. Montault, writing about the time of the Paris epidemic (1828–1830), quotes Francois as having recognized the same disease (Paris epidemic) in the West Indies among the negroes, who lived in houses in low and damp places.

In 1888 Dr. Henry Strachan, senior medical officer, reported 510 cases of "Malarial multiple neuritis" observed in the Kingston (Jamaica) Public Hospital, full notes having been taken on 121 of these cases. He described the symptoms in great detail, and they are the same as described by the French authors as acrodynia. Both Sir Patrick Manson and Dr. F. M. Sandwith studied Strachan's cases and neither agreed with his diagnosis and the latter suggested that the disease was acrodynia. A recent communication (February 19, 1923) received from Dr. G. T. Hunt, Medical Officer at Kingston, Jamaica, says, "The cases still prevail in this colony of the type reported on by Dr. H. Strachan, who was at one time Senior Medical Officer of the Public Hospital, Kingston, Jamaica."

Acrodynia has been recognized in Mexico for many years.

Effort has been made to ascertain whether it has occurred in South America but so far no convincing evidence has come to our attention.

We suspect that the disease is present in India. Dr. Evers (British Medical Service) in an article published in the Indian Gazette, August 2, 1880, describes a disease in which the chief manifestation is intense burning of the feet. He says that this disease is common in India. He calls it "Ignipedites."

Reports have come from Turkey, Persia and Algiers of unmistakable cases of acrodynia.

Within the last three years articles have appeared from Canadians that lead us to believe that acrodynia has existed in Canada for more than 20 years and that for the last few years cases have been occurring with increasing frequency.

In 1922 Dr. A. F. McKenzie ofAlliston, Ont., read a paper before the meeting of the Ontario Medical Association, entitled "Acrodynia. A Report of Three Cases in Rural Practice." The first of these cases, Mary N. aged five years, was seen first on February 14, 1899. The second Kate H., was first seen November 27, 1899, the third, Baby N., aged seven months, was first seen March 18, 1901.

These cases have been carefully studied and they follow very closely the descriptions of those reported in the Paris epidemic and the sporadic cases that for many years appeared throughout France and other countries of continental Europe.

There was published in the Archives of Pediatrics, Vol. xxxviii, No. 10, October, 1921, a most interesting and valuable article entitled

"A Clinical and Metabolic Study of Acrodynia" by Alan Brown, M.B., Angelia M. Courtney, A.B. and Ida F. MacLachlan, B.A., of Toronto.

They report eight cases, two having been seen in 1918 and considered more or less like *pellagra.* "But after consultation with Professor Andrew Hunter, who had become familiar with pellagra as biochemist at the Pellagra Hospital established by the U. S. Public Health Service, this condition we excluded."

A most interesting and characteristic situation has arisen in Australia, one not unlike the situation that existed in Europe before acrodynia obtained a definite status. Dr. H. Swift, of Adelaide, read a paper before the Australian Medical Congress at Auckland, New Zealand, in 1914. His subject was "Erythroedema."

He reported 14 patients ranging in age from 6 months to 15 months. He gave an excellent general description and it may be added that they conform in detail to many of those described by the French and other writers on acrodynia already referred to. Following Dr. Swift's paper was that of Dr. A. Jeffreys Wood of Melbourne, published in the Medical Journal of Australia, February 19, 1921. He gave a clear and vivid picture of his cases, also a brief historical sketch which is both interesting and illuminating. It is as follows: "To Dr. Swift of Adelaide, belongs credit of having been the first medical man to describe this condition as it exists in children. His paper read at the tenth session of the Australian Medical Congress at Auckland, New Zealand, in 1914, was the first attempt to tabulate the peculiar sets of symptoms that he has grouped together under the term 'erythroedema.'

"Being interested in his paper I wrote to Dr. Swift to ask him where he got the name for the disease; he replied that he had invented it himself to convey the picture of redness and swelling of the hands and feet, but was not altogether satisfied with it as there was no real edema, pitting being absent.

"I have frequently seen cases in Melbourne for the past 30 years, and before my time Dr. William Snowball, of the Children's Hospital, Melbourne, recognized the complaint and spoke of the children with the 'raw beef hands and feet.' He was of the opinion that the symptoms frequently followed severe attacks of gastroenteritis and that the prognosis was generally good. In Sydney this illness has long been recognized and it is generally spoken of by Dr. Clubbe and others as 'the pink disease.'

"Since hearing Dr. Swift's paper in Adelaide I have been much interested in collecting notes of these cases; I now have notes of 40 cases and Dr. F. Hobill Cole has notes of 51 cases, most of them having been collected since Dr. Swift's paper became available about 1917. So that when the disease is recognized it is evidently not an uncommon one."

Just how long acrodynia has existed in the United States is impossible to even estimate. Some who have had a rather extensive experience with pellagra, and have since become familiar with the symptoms characteristic of the several types of acrodynia recall cases of so-called atypical pellagra that would at this time be classified as cases of acrodynia. To classify such cases would be difficult, if not impossible.

The first report we have been able to find in American medical literature is by Dr. W. J. Cole of Blairsville, Indiana, who published in the St. Louis (Mo.) Medical and Surgical Journal of April 5, 1880, three cases of intense burning of the hands which occurred in his practice during three years. Having read the reports of the Paris epidemic, he pronounced them acrodynia. Dr. Cole's report was so brief that an investigation was undertaken in order that it might be supplemented in case he could be located. He was finally located in Larkin, Florida, where he is living in retirement. He wrote that he had kept no notes of the cases and whatever information he could furnish would be from memory. He has written two interesting and courteous letters in reply to those he received, in neither of which is contained evidence of such a character as would justify the responsibility of confirming a diagnosis.

To Dr. J. B. Bilderback of Portland, Oregon, is due the credit of being the first in the United States to have recognized and reported a series of cases of acrodynia, that had come under his observation. These were reported at a meeting of the North-Pacific Pediatric Society, Seattle, Washington, January 17, 1920, in an article entitled "A Group of Cases of Unknown Etiology and Diagnosis." Dr. Bilderback reported ten cases, the first having been seen in the spring of 1914. Dr. Bilderback's report was followed by a paper before the Section on the Diseases of Children, A. M. A. at New Orleans, April, 1920, by William Weston, M.D. entitled "Acrodynia." This paper reviewed Dr. Bilderback's cases, and acknowledged his priority of description and undertook to identify them with acrodynia as described by the French writers.

Following this paper there appeared in the November 1920, number of the American Journal of Diseases of Children an article by Dr. Albert H. Byfield of Iowa City, entitled "A Polyneuritic Syndrome Resembling Pellagra-Acrodynia (?) Seen in Very Young Children." Dr. Byfield reports having seen seventeen cases within five years, all of them under four years of age. An excellent description is given and it conforms to those given by the French authors.

Following these articles were numerous others reporting cases from every section of the United States. Reference must be made to a series collected by Dr. Frank H. Lamb of Cincinnati. It has been our privilege to have a copy of these case reports and they are the most interesting so far collected in the United States. They illustrate most of the types discussed in the French literature.

Medical men are familiar with the fact that the American and French opinions about the cases coincide. The history of acrodynia is the same in every country where it has occurred. Readers are referred to the original French articles which were written with such infinite care and attention to detail. The accounts by Delmas and Valleix will be found most valuable.

Etiology.—The cause of acrodynia is not known. There have been many hypotheses advanced at different times. Most frequently it has been associated with some other disease. In the very old literature acrodynia is confused with pellagra. In fact, practically all the German writers felt that it was a peculiar and an unusual form of pellagra and thought it was caught by the ingestion of spoiled grain. This idea was at first predominant among the French writers. Further investigation caused them to separate the two diseases. However, this agreement as to the two diseases being distinct did not result in the complete abandonment of the spoiled grain theory as all the writers mentioned it as a possible cause. Other causes were sought for by observers in France and other countries, and among the more prominent suggested were: Food deficiency, arsenic poisoning and as a terminal stage of some other disease. The disease most often suspected of relationship in some way with acrodynia is influenza. It is interesting to note that each of these hypotheses are advocated by some at this time.

It is difficult to reconcile any of the hypotheses mentioned with the accounts of the Paris epidemic. We have seen that in certain barracks practically the entire population became ill with this malady, while others escaped entirely, although the rations were the same in all. The disease seems to have spread from one neighborhood to another, thence over Europe and other parts of the world in the same manner as had other great epidemics that we now know were of an infectious nature. This fact alone would seem sufficient to discredit food deficiency or arsenic poisoning as causative factors.

From careful investigation it appears that the great influenza epidemic so often referred to by writers who undertake to establish an etiologic relationship between acrodynia and influenza did not occur occur until 1832, or not until two years after the acrodynia epidemic had spent its force.

Some recent American writers are inclined to emphasize the fact that acrodynia is coincident with or immediately follows an epidemic of influenza. They might also have directed attention to the fact that both the influenza and epidemic cerebrospinal meningitis prevailed at about the same time in the same localities in pandemic proportions, and that each was unusually severe. Yet no one would claim that one was either the cause or result of the other. Acrodynia, like influenza and cerebrospinal meningitis are often observed as occurring sporadically in the same communities and this fact has no etiologic significance.

There has recently been advanced the theory that acrodynia was due to an endocrine disturbance (adrenal).

Some of the more recent writers have called attention to the possibility of acrodynia being caused by focal infections. This has not been confirmed. There were several observations made by the early writers that time has sustained. There does not appear to be any hereditary influence; no age is exempt. Infants are as susceptible to the disease as adults. Hygienic and sanitary conditions seem to have no etiologic influence. Season and climate have no etiologic bearing. It may be stated that the disease is not contagious.

Diagnosis.—A well advanced case of acrodynia once seen will never be forgotten or mistaken for any other disease.

The disease most likely to be mistaken for acrodynia is pellagra.

The following table of differential diagnosis is presented.

ACRODYNIA.	PELLAGRA
Occurs at any season.	Usually commences in spring or autumn.
No insanity.	Insanity often present.
No tendency to recurrence.	Recurrence usual.
Rash most pronounced at ends of fingers gradually fading until it disappears above wrists.	Symmetrical rash with sharp line of demarcation on forearms and legs. No tendency to fade.
Dorsal surface of feet and ankles seldom involved.	Dorsal surface of feet and ankles usually involved.
Erythematous rash may be present over the entire body with the exception of back of neck.	Not observed in pellagra.
Palms and soles the seat of intense burning, itching or perhaps numbness.	Not present in pellagra.
Diarrhea not usually serious.	Obstinate diarrhea often present.
Uncomplicated cases invariably recover.	Mortality comparatively high.
Photophobia present in two-thirds of cases.	Photophobia usually absent
Great wretchedness in nearly all cases.	Wretchedness seldom occurs.
Absence of appetite the rule.	Not usually impaired in children.

Symptoms.—In reviewing reports of cases from this and other countries we are impressed with the observation of French writers that we may expect variations in symptoms not only in different localities but in the same locality.

The onset of the disease may be either abrupt or so gradual that its true nature may not become manifest for several weeks. The child becomes peevish and fretful, loses his appetite and sleeps poorly. Soon the feet and hands become red and swollen accompanied by intense itching or burning of the palms and soles. A rash may also appear on the tip of the nose, the ears and cheeks or it may cover the entire body except the back of the neck. Soon after the rash appears, desquamation begins. The desquamation sometimes involves all the layers of the skin and deep necrotic ulcers form. Usually, however, it only involves the superficial layers, leaving a bluish red glistening surface, giving a cold and clammy sensation to the touch. These areas may be either hyperesthetic or anesthetic. Coincident with the symptoms just enumerated profuse irritating sweats occur that add to the child's discomfort. The eyes become irritated and very sensitive to light. The patient becomes pale and emaciated. The hair becomes dry and thin and areas of alopecia appear. The muscles become flabby and if he has walked he can no longer do so. He lies with face buried in the pillow or against his mother's shoulder, his hands in constant motion, feet rubbing against each other and constantly moaning or begging that his feet be rubbed. Such a picture of abject misery once seen can never be forgotten.

Skin.—The skin manifestations are uniformly present and invariably involve the palms and dorsal surface of the hands and soles, less often the dorsal surface of the feet. They are cold, clammy and cyanotic. There occurs an erythematous rash most marked at the tips of the fingers and gradually diminishes until at the wrists or forearms it entirely disappears. There is no line of demarcation as in pellagra.

Less often there occurs a rash on the tip of the nose, the ears and cheeks. Occasionally the entire body except the neck, is covered with an erythematous rash. It may be constant or disappear and recur.

When it disappears, pigmented spots occur. As the rash fades there will be seen a desquamation which may involve only the superficial layers of the skin or may involve all layers. The surface is bathed in a profuse, irritating sweat. The extremities are invariably edematous, but there is no pitting upon pressure. Less often this edema may be observed about the face or other parts of the body. The eruption usually appears as an erythema, but may present itself as macules, papules or pustules and in a given case all may be observed. The trophic changes in the skin may at times be very severe and result in gangrene of the fingers or deep ulcers on the toes or soles.

Hair.—The hair is dry and tends to fall out. It not infrequently happens that the child pulls out "handfuls." In other cases he will pull out hair by hair leaving bald spots.

Ears.—Otitis media and mastoiditis have been observed.

Eyes.—Photophobia with conjunctivitis, and lachrimation are the rule.

Sometimes injections of the cornea and keratitis are present.

Teeth.—Loss of teeth with or without pathologic changes in the gums is a manifestation often observed.

Nails.—Change in the color of the nails is of frequent occurrence. Shedding of the finger and toe nails occurs in many cases.

Genito-urinary.—Pyelitis accompanied by frequent and painful micturition and irregular fever is met with in many cases. Both albumin and acetone are found occasionally.

Gastro-intestinal.—Loss of appetite is common. Anorexia is often present. Diarrhea and vomiting are met with in a small proportion of cases. Prolapse of the rectum has been occasionally reported. It may be a severe and troublesome symptom.

Nervous System.—Irritability, restlessness, insomnia and trichotillomania occur in nearly all cases. Paresthesia of the fingers, hands, toes and feet, especially the palms and soles is invariably present.

Convulsions occur in a few cases.

Glandular.—Cervical glands often enlarged.

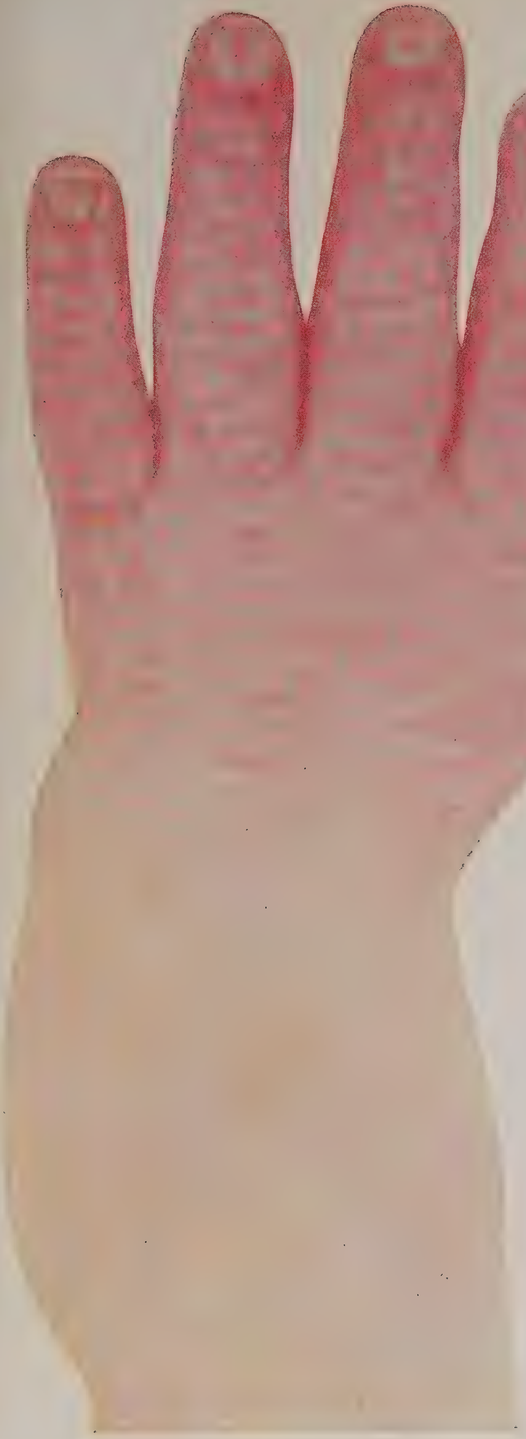

Fig 179.

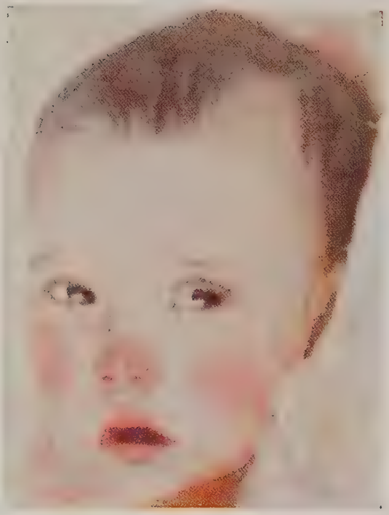

Fig. 180.

Figs. 179, 180.—Acrodynia (Dr. Albert H. Byfield, in American Journal of Diseases of Children).

Fig. 179.—Eruption on the hands with characteristic tendency to fading out toward the wrist

Fig. 180.—Same case as Fig. 179. Involvement of cheeks and nose. This was the only case in which an eruption of any intensity appeared on the face.

Reflexes.—The reflexes may be unaltered, increased, diminished or abolished.

Muscular.—There occurs a distinct loss of tone in all the muscles. They become flabby and atrophy. Sometimes marked contractions take place.

Respiratory.—A nasopharyngitis is usually present. This occasionally develops into a bronchitis or a bronchopneumonia. A pulmonary edema occurs.

Circulatory and Blood.—The heart rarely shows any change. A secondary anemia is present in a fairly large proportion of cases.

Temperature.—A slight rise in temperature often occurs. High temperature is seldom met with unless due to some complication.

Laboratory Findings.—There usually exists a fairly regular relative polymorphonucleosis and an increase in the total number of leukocytes.

The red cell count is not materially altered. The Wassermann is uniformly negative.

Spinal fluid: Negative.

Tuberculin skin tests and Schick tests negative.

Blood cultures uniformly negative.

Feces negative for parasites.

In cases studied from a metabolic standpoint it was observed that there was a lowered or negative nitrogen balance in some and in others a negative balance of bases. The excessive loss of nitrogen and bases occurred through the urine.

Prognosis.—The outlook for the recovery is usually good unless some complication takes place. Bronchopneumonia and pulmonary edema may produce fatal results.

There is usually a long, trying period of invalidism in practically all cases of acrodynia.

Pathology.—Autopsies on cases have uniformly developed the fact that death was due to a complication rather than to acrodynia.

Treatment.—The most serious problems encountered in the treatment of these cases are: (*a*) Feeding, (*b*) Relief from pain, (*c*) Insomnia, (*d*) Anorexia.

The first indication should be met as in any difficult feeding case, and if necessary, food should be administered by gavage.

Relief from pain is most successfully accomplished by frequent applications of cold calamine lotion to all irritated surfaces. It is our experience that opiates, bromides and chloral are of little or no avail.

Insomnia is largely dependent upon the paresthesias. Relief from these symptoms may be obtained by the method already suggested.

Anorexia is best treated by giving small quantities of food, frequently repeated. Thick cereals, fruit juices and substances containing the fat soluble and water soluble vitamines should be offered.

No results have been obtained by the use of the glands of internal secretion.

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